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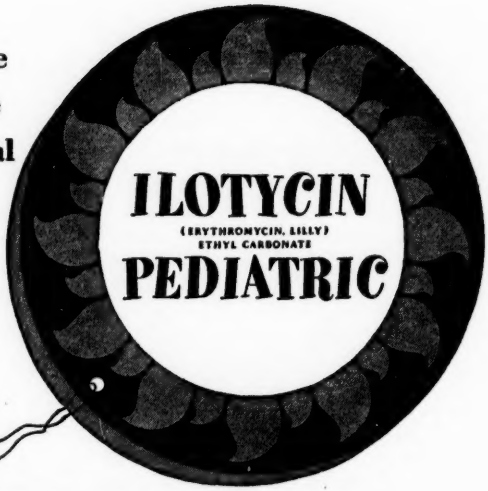

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Is Asphyxia a Pathological Entity Recognizable Post Mortem?

Although 'asphyxia' means 'pulselessness', the term has come to mean, in medico-legal usage, mechanical interference with the processes of breathing. Modern developments in the physiology of tissue respiration have broadened the concept of asphyxia, but for most practical purposes the term connotes the mechanical type of interference mentioned.

It is also frequently alleged that signs can be seen in the dead body which provide conclusive evidence that death in a particular case was in fact due to asphyxia, as this term is commonly understood. Indeed, some authors take a similar view about the diagnosis of death due to coma and even claim to be able to recognize, from the autopsy findings, a 'mixed' form of death described as 'comato-asphyxia'.[1]

In the diagnosis of asphyxia after death has taken place, reliance is commonly placed on the following signs: congestion of internal organs, petechial haemorrhages throughout the body, cyanosis, fluidity of the blood and dilatation of the heart. Modern knowledge has made it clear that congestion of internal organs as well as petechial haemorrhages are not diagnostic of asphyxia; and cyanosis is a term which should be confined to the living because there is no way of distinguishing it from the post-mortem lividity (hypostasis) which is inevitable after death. The work of Mole[2] and Ponka and Lam[3] has demonstrated conclusively that the fluidity of the blood in a dead body bears no conceivable relationship to an asphyxial mode of death. Dilatation of the heart is also of no pathognomic significance.

1. Glaister, J. (1949): *Medical Jurisprudence and Toxicology*, 9th ed., p. 145. Edinburgh: E. & S. Livingstone Ltd.
2. Mole, R. H. (1948): *Fibrinolysin and the Fluidity of the Blood Post Mortem*, J. Path. Bact., **60**, 413.
3. Ponka, J. L. and Lam, C. R. (1948): *Effect of Asphyxia on Blood Coagulation in Dogs*, Proc. Soc. Exp. Biol. Med., N.Y., **68**, 334.

As all the signs commonly relied upon for establishing asphyxia are non-specific, i.e. not peculiar to asphyxia, and as all these signs are also found commonly in many deaths from natural causes, the presence of these signs loses all diagnostic value. It is possible, however, that the topographical distribution of, e.g. petechial haemorrhages, may suggest that mechanical obstruction to the venous return preceded and was the cause of death (as in strangling or throttling); but in such cases it is necessary to appreciate that these haemorrhages (if other causes can be excluded) have the same significance as the finger marks on the neck in throttling, an associated fracture of the hyoid bone, the ligature mark round the neck in strangling, the special signs associated with drowning, or the colour of the blood in carbon monoxide or potassium cyanide poisoning. However, considerable care must be taken in evaluating what appear at first sight to be the results of mechanical obstruction to the venous return from the head and neck, since marked engorgement of the head and neck (with petechial haemorrhages in the conjunctivae) may occur in a variety of deaths ranging from coronary thrombosis to head injuries. Nor must we disregard the gravitational effects occurring *post mortem*, which may influence the morbid anatomical picture.

In drawing attention to the need to distinguish between the basic pathological signs common to almost all deaths, however initiated, and the specific signs to be differentiated from them, Gordon[4] introduced a valuable concept which should have a profound and important influence on modern forensic practice. His hypothesis underlying the mechanism of modes of deaths of medico-legal importance can, of course, be applied to other forms of death than those due to asphyxia in the commonly accepted sense.

Asphyxia is probably a term which has outlived its scientific usefulness. In so far as it conveys the concept of deprivation of an essential oxygen supply, it could better be replaced by the modern physiological term 'anoxia'. Anoxia, in turn, can be qualified,[4] as physiologists have already done in their own field, to classify 4 major groups of death:

i. Anoxic anoxia (e.g. mechanical interference with the respiratory tract);
ii. Anaemic anoxia (e.g. haemorrhage);
iii. Stagnant anoxia (e.g. shock);
iv. Histotoxic anoxia (e.g. potassium cyanide poisoning).

On the basis of this classification it is clear that more often than not the pathologist is only in a position to say that his findings are *consistent* with asphyxia in the sense in which this term is commonly used in Court practice.

This limitation on scientific knowledge at the present time must be stressed in view of the lamentable tendency, strongly to be deprecated, which expects that the pathologist who has examined a dead body is necessarily in a position to indicate precisely to the Court what the cause of death was. A

4. Gordon, I. (1944): *A Classification of Deaths of Medico-Legal Importance*, Brit. Med. J., **2**, 337.

dispassionate survey of the data makes it clear that this is not always possible, especially in cases of so-called asphyxia, where the findings may need to be considered with *all* the other evidence before an opinion can be ventured. Autopsy findings may well not be of *positive* assistance. The value of *negative* findings may be of crucial importance, as Adelson has recently pointed out in connexion with a cognate problem.[5]

Much of the unwarranted dogmatism that pervades this branch of pathology is due to the confusion that results from the uncritical transfer of clinical observations appropriate to the living, to describe a different order of things in the dead. Coma, syncope, asphyxia and cyanosis, e.g. are bedside observations inappropriate and misleading when applied to the dead. This distinction is implicit in Gordon's hypothesis, and provides a necessary discipline and corrective to our thinking about problems in this field.

This important issue has been restated very clearly in the third edition of Professor Gordon's[6] textbook reviewed elsewhere in this issue. His thoughtful contribution more than justifies the publication of yet another textbook of forensic medicine and it is certain that his views in this field will not only receive very serious consideration by medico-legal pathologists all over the world, but will also strongly modify their practice.

5. Adelson, L. (1953): *Possible Neurological Mechanisms Responsible for Sudden Death with Minimal Anatomical Findings*, J. Forensic Med., **1**, 39.
6. Gordon, I., Turner, R. and Price, T. W. (1953): *Medical Jurisprudence*, 3rd ed., Edinburgh: E. & S. Livingstone Ltd.

Death from Dicoumarol Poisoning

The Case of Regina *vs.* T. v d M.*

The Indictment and the Verdict

A medical practitioner was tried before the Supreme Court of South Africa for culpable homicide on an indictment which alleged that he had caused the death of the deceased, his patient, by his negligent acts or omissions. The particulars of the negligence relied on by the Crown were specified as follows:

That the accused, in prescribing and administering to the deceased a preparation known as dicumarol, failed to exercise that degree of skill, care, diligence and ability which he could reasonably be expected to exercise, in that:

(*a*) He failed, before administering and prescribing the said preparation, to acquaint himself with the various strengths or dosages in which the preparation is dispensed;

(*b*) He failed to specify on the prescription written out by him, the strength or dosage of the preparation to be dispensed to the deceased;

(*c*) He administered the said preparation without any, or adequate, knowledge of its effects and of its proper administration;

(*d*) He failed, when prescribing the said preparation, to inform the deceased, or her husband, of the potentially toxic nature of the drug, of the precautions to be observed, and of the necessity of noting and reporting any signs of abnormal bleeding;

(*e*) He failed, when administering the said preparation, himself to keep the deceased under observation, or to arrange for her to be kept under proper observation for signs of bleeding or other effects;

(*f*) He failed, in administering the preparation to the deceased, to control the dosage by means of the guidance afforded by prothrombin index tests;

* In the Supreme Court of South Africa (Witwatersrand Local Division), Johannesburg, 20 May 1953.

In the Judge's *Summing Up* the names of the accused, the deceased and her relatives have been abbreviated to initials in the interests of anonymity; references to the cases mentioned by Mr. Justice Roper have been inserted.

We are indebted to Mr. N. C. Masters, B.A., LL.B., Palace of Justice, Pretoria, for the preparation of the legal summary of the case report and to Dr. J. Friedman, Senior District Surgeon, Johannesburg, for the medical summary.

Differing national, proprietary and judicial usages have made uniform spelling of Dicoumarol impossible.

The *Summing Up* is reproduced by permission of the Editor and the Publishers of the *South African Law Reports*.—*Editor.*

(g) He failed to take proper and adequate measures on being advised that the deceased showed signs of abnormal bleeding.

The jury brought in a unanimous finding that the accused was guilty of the crime as charged. A fine of £75 (seventy five pounds) or three months compulsory imprisonment was imposed by the Judge.

The Facts

On 29 July 1952 the deceased, a married female 70 years of age, consulted the accused in his capacity as a medical practitioner carrying on a general practice in Pretoria. The deceased gave a history of an old 'white leg' on the left side and of having had a femoral thrombosis there recently which still gave trouble and which had apparently not cleared up. The examination by the accused revealed that some activity as regards thrombosis was still present. He decided to treat the condition with dicumarol, and accordingly gave to the deceased the following prescription:

'Mrs. . . . .
Dicumarol, 40 tabs
One t.d.s. p.c.
Sgd.

The chemist to whom the prescription was handed, being unsuccessful in his attempt to communicate with the accused, consulted the British Pharmacopoeia and dispensed 40 tablets of 100 mg. strength each.

The deceased, who (to the accused's knowledge) was returning to Johannesburg the following day, commenced immediately to take the tablets, and continued to do so until 10 August. In all she took 38 tablets or 3,800 mg. of dicumarol in 13 days.

The first signs of haemorrhage appear to have manifested themselves 8 days later (5 August) when the daughter noticed there were little black spots on her mother's leg, like blisters, and the leg was a little redder than the other. Three days later (8 August) her gums were bleeding and there was bleeding from a sore between the shoulder. The accused, when advised by telephone of her condition on 10 August, stopped the tablets, but did not visit the patient or prescribe any treatment.

From then on her condition deteriorated. There was continuous nose bleeding, bleeding from the bladder and from the mouth, and she seemed to be passing urine more frequently than normal. These symptoms were reported to the accused by the husband on 13 August and 'Vitamin K, 10 mm.' was ordered to be given. No improvement was observed, and on 15 August at the request of the husband, the accused himself being unable to visit the patient, a local general practitioner was called in. He arranged for the deceased to be moved to a nursing home the same day where she was later seen by a specialist. Despite transfusions of fresh blood and vitamin K injections no recovery took place and death occurred on 21 August.

The Defence

The Defence did not dispute that the generalized haemorrhage, which caused death, was due to dicumarol poisoning; while the accused, in giving evidence, admitted that he was not aware of the potential dangers of the preparation or of the special precautions to be observed in its administration.

The Defence rested almost entirely on the plea that the proximate cause of the deceased's death was the negligence of the chemist in dispensing an improper or incomplete prescription without first communicating with the practitioner who had issued it. In elaboration of this plea, the accused stated that some 2 or 3 years before he had become acquainted with the preparation dicumarol as the result of a conversation between colleagues. He remembered from this conversation that a dosage of 25 mg. 3 times a day was mentioned, and that it was not a dangerous preparation. He admitted that this was the only knowledge he had of dicumarol when he prescribed for the deceased, and it explained why he had not inserted on the prescription the strength of the tablets to be dispensed. Had the chemist consulted him, the fact that the tablets were made up in tablets of strengths other than 25 mg. would have been brought to his notice and the fatal overdosage thus avoided.

Medical Evidence

Clinical Findings. The specialist who examined the deceased in the nursing home on 18 August found the patient conscious, rational and co-operative. She was not anaemic clinically, and only complained of severe headache. She was febrile.

Her pulse rate was 90 per minute, and the blood pressure 140/60 mm. Hg. The pupils were very contracted. There were no cranial nerve lesions. Slight nuchal rigidity. Sternal tenderness. No paresis or paralysis. The fundus of the eye was difficult to examine because of the small pupils, but there was some swelling of the right disc. No retinal haemorrhages were seen. The upper limb reflexes were present and equal; knee and ankle reflexes were absent as well as the abdominal reflexes. The left leg was slightly swollen, with the veins prominent. There was no sign of active phlebitis. There were several haemorrhages into the skin of the back and there were large bruises over the buttocks. Multiple petechial spots were present over both legs and no haemorrhages were seen in the mouth. The heart was not enlarged. No murmurs were heard. The lungs were clear; the abdomen not abnormal; no liver or splenic enlargement was detected. The urine was dark red in colour, apparently blood stained.

On 19 August continued bleeding from the renal tract was noted; also an increase of headache and some clouding of consciousness. Abdominal distension appeared.

On 20 August there was marked impairment of consciousness, with probable paresis of the right arm, and marked abdominal distension.

On 21 August (the date of death) the patient was comatose. The pupils were dilated and not reacting to light. There was bilateral papilloedema, with paresis of the right arm. The patient vomited blood.

Treatment. Transfusions of fresh blood and vitamin K were given after the deceased's admission to the Nursing Home on 15 August.

Prothrombin Estimations. On 15 August the test showed that the plasma coagulated at widely varying times ranging from 42 to 100 seconds. It was therefore not possible to give an estimate of the prothrombin index. On 16 August the prothrombin index was 37%. On 19 August the test plasma failed to coagulate in 2 minutes and no estimate was therefore possible. On 20 August the index was estimated at 34%, and on 21 August at 27%.

Toxicological Findings. The viscera were submitted for analysis on 27 August and a weak positive reaction for dicumarol was obtained.

Autopsy Findings. The cause of death was given as generalized haemorrhage associated with hypoprothrombinaemia, and the following observations were noted:

'The body was that of an average developed elderly European female, 66 inches in length and weighing 120 lb. There were multiple large confluent ecchymotic areas on both upper limbs, both buttocks and both thighs. The lips and conjunctivae were pale. Needle puncture marks were present in both thighs and the right antecubital fossa. A lumbar puncture mark was present.

The scalp vessels were engorged. There were multiple small subaponeurotic haemorrhages into the frontal region of the scalp. The scalp revealed no evidence of injury. There were present a most massive generalized fresh subdural haemorrhage with resultant compression of the cerebral convolutions. Marked elongation of the cerebellar tonsils and bilateral uncus herniation were observed. There was extensive haemorrhage into the substance of both lobes of the cerebellum and the fourth ventricle was filled with blood. There was softening of both lobes of the cerebellum in the region of the haemorrhage. No satellite contusional haemorrhages could be detected, nor were there contusional haemorrhages anywhere else throughout the brain substance.

There was a moderate collection of blood within the subarachnoid space localized to both occipital poles. The lateral ventricles were markedly dilated, there was increase of cerebrospinal fluid in the subarachnoid space and the brain was congested.

The cerebral arteries were healthy and revealed no signs of atheroma and there was no evidence of a ruptured congenital aneurysm.

No lacerations of the meninges or fractures of the bones of the skull could be found.

The heart weighed 11 oz. The valvular structures were normal and healthy. The coronary arteries were patent and healthy and revealed no atherosclerosis. A slight increase of epicardial fat was observed and there was a small subendocardial haemorrhage into the interventricular left septum. The heart muscle was pale and flabby and there was a small amount of atheroma of the aorta. The thyroid gland was normal and revealed no haemorrhage within its substance.

The lungs were adherent to the parietal pleura. They were congested and oedematous and there were haemorrhagic areas in the lung substance. The right lung weighed 19 oz. and the left lung weighed 17 oz. The liver surface was smooth. On section the liver showed some congestion and there were multiple discrete areas of fatty degeneration throughout. The gall bladder contained a single small cholesterol calculus the size of a pea and there was no evidence of biliary obstruction. The spleen was soft and congested and there was some increase of lymphoid tissue.

There was massive perivesical, retroperitoneal and bilateral perinephric haemorrhage present. The kidney capsules stripped easily revealing multiple small subcapsular haemorrhages. The kidney surfaces were finely granular and scarred. There were large submucous haemorrhages into both kidney pelves and the adjacent medullary tissues. There was free blood in both kidney pelves.

The suprarenal glands were normal.

The bladder was distended and filled with coffee-coloured smoky urine. There was submucous haemorrhage into the upper half of both ureters.

The uterus and adnexa were normal.

There were multiple petechial haemorrhages into the gastric mucosa. The gastro-intestinal system was otherwise normal. A large peri-rectal haemorrhage was observed. The pancreas was normal.

From the Judge's Summing Up

Roper, J.: Gentlemen of the jury, as you have seen in the indictment before you, the accused is charged with the crime of culpable homicide. It is alleged that in the period between the 29th July and the 15th August, 1952, he wrongfully and unlawfully killed C. M. B. He has pleaded not guilty. You have heard the evidence adduced by the Crown and by the defence, and it is for you to say whether he is guilty or not of that charge . . .

Now, the accused, as already mentioned, is charged with culpable homicide, which may be defined as the unlawful killing of a human being without intent to kill. One way of killing a person unlawfully is to do so by negligence, as, for instance, killing a person by driving a motor car negligently. In the present case the Crown suggests another form of negligence with which I will deal presently.

First of all, what is negligence? It is simply this, gentlemen. A person is negligent if he fails to exercise that degree of care which a reasonable man would exercise in the particular circumstances. That is a definition which applies to all forms of negligence, and the definition has a somewhat special application in the case of a member of a skilled profession, such as a doctor, because a man who practises a profession which requires skill holds himself out as possessing the necessary skill, and he undertakes to perform the services required of him with reasonable skill and ability. That is what is expected of him and that is what he undertakes, and, therefore, he is expected to possess a degree of skill which corresponds to the ordinary level of skill in the profession to which he belongs. In deciding whether a member of a profession, such as a doctor, is negligent or not the question is whether applying the general definition I have mentioned, he has exercised the degree of care and skill which a reasonable man, who is also skilled in the profession, would employ. That is the test in the case of a doctor. Although a medical practitioner is not expected to bring to bear, in the case entrusted to him, the highest possible degree of professional skill, he is bound to employ reasonable skill and care—not the highest, not the specialist's skill, but reasonable skill and care. In deciding what is reasonable regard must be had to the general level of skill and diligence possessed and exercised by the members of the branch of the profession to which the practitioner belongs. The standard is the reasonable care, skill and diligence which is ordinarily exercised in the profession generally. That statement of the law is taken from a number of decisions of our Courts in cases in which the duties of

medical practitioners have been considered, and I will not weary you with those cases.[1] I repeat, the standard required by a doctor is that reasonable care, skill, diligence and knowledge which is ordinarily exercised by the profession generally.

There is a passage in one case which I want to read to you, gentlemen, because it seems to me to set out what is required of a medical practitioner in relation more particularly to criminal cases. This is the case of *The King v. Bateman* (133, Law Times Reports, 780). It is a case that came before the Court of Criminal Appeal in England, and the judgment of the Court was delivered by Lord Chief Justice Hewart. After referring to a number of English decisions on this branch of the law, he said that the law might be summarised in this way:

'If a person holds himself out as possessing special skill and knowledge and he is consulted as possessing such skill and knowledge by or on behalf of a patient he owes a duty to the patient to use due caution in undertaking the treatment. If he accepts the responsibility and undertakes the treatment and the patient submits to his direction and treatment accordingly he owes a duty to the patient to use diligence, care, knowledge, skill and caution in administering the treatment. No contractual relation is necessary nor is it necessary that the service be rendered for reward. It is for the Judge to direct the jury what standard to apply and for the jury to say whether that standard has been reached. The jury should not exact the highest or a very high standard nor should they be content with a very low standard. The law requires a fair and reasonable standard of care and competence. The standard must be reached in all the matters above mentioned',—that is, diligence, care, skill, knowledge and caution—'If the patient's death has been caused by the defendant's ignorance or carelessness, it will not avail to show that he had sufficient knowledge, nor would it avail to prove that he was diligent in attendance if the patient has been killed by his gross ignorance and unskilfulness. No further observation need be made with regard to cases where the death is alleged to have been caused by indolence or carelessness. As regards cases where incompetence is alleged it is only necessary to say that the unqualified practitioner cannot claim to be measured by any lower standard than that which is applied to a qualified man.'

It means, gentlemen, that a practitioner in a skilled profession cannot shelter himself behind a defence that he did know enough about it or was not sufficiently skilled. I have read that general statement as to the duties of medical practitioners and the degree of care and skill which is required of them because it seems to me to set out the position admirably.

There is one phase in particular of a medical practitioner's duties to which I ought to refer, and that relates to a practitioner's use of a drug with which he is unfamiliar. It seems to me a matter of common sense that a doctor should not use a drug that is new or unfamiliar to him without satisfying himself as to its properties, as to the possibility of it being dangerous, or without acquainting himself with the proper methods of using it. In a case,[2] which was referred to by counsel for the Crown yesterday, this was laid down—I am referring just to the headnote:

[1] For example, Mitchell *vs* Dixon, 1914 A.D., 519; van Wyk *vs* Lewis, 1924 A.D., 438; Coppen *vs* Impey, 1916 C.P.D., 309.

[2] R *vs* Chamberlain (1867) 10 Cox, C.C. 486.

'There must be a competent knowledge and care in dealing with a dangerous drug. If a person was ignorant of the nature of the drug he used, or was guilty of gross want of care in the use of it, he would be criminally responsible for the consequences'.

The facts in that case were that the accused, who was a herbalist, administered and sold an ointment to a woman who had a tumour, with the result that she died. There was no warning as to the dangerous nature of the ointment or any directions as to the use of it. He was found guilty of culpable homicide. Another case[3] was quoted to you yesterday by counsel for the Crown, where a young practitioner in this country had treated patients with a preparation of arsenic. He had been told by a colleague that an ampoule of the drug contained one dose and that he should use it accordingly. It turned out that each of the ampoules he administered contained the equivalent of ten doses. The result was the death of two patients to whom the full contents of an ampoule were given. The fact that the ampoule contained ten doses would have been apparent to this young man if he had read the instructions printed upon the ampoules, but he did not do so. It was held that he was negligent and he was accordingly convicted of culpable homicide. These cases show, gentlemen, that before a medical practitioner uses a dangerous drug with which he is unfamiliar he must satisfy himself as to the properties of the drug, and he cannot defend himself, if he is called to account afterwards, by saying that he did not know; it is his duty to know.

Now, I must say something about the difference between specialists and general practitioners, since there is a recognised difference in the medical profession. In this case evidence has been given by several specialists, who have told you, among other things, what they would do in certain circumstances. I must warn you that, when a general practitioner is tried, the test is not what a specialist would or would not do in the circumstances, because a general practitioner is not expected to have the same degree of knowledge and skill and experience as a specialist has. When a specialist tells you that he would do this, that or the other thing it does not follow that you must expect the general practitioner to act in the same way. But the question is, what is the common knowledge in the branch of the profession to which the accused belongs? What is common knowledge and accepted practice among general practitioners? When the specialists tell you what is common knowledge in the profession, then that is evidence which you are entitled to rely on, because the general practitioner is expected to be possessed of knowledge which is common in the profession. When the specialists tell you of the dangerous properties of the drug with which this case is concerned and when they tell you that that is common knowledge in the profession, then that is evidence which you are entitled to accept.

In the present case the Crown alleges that the accused caused the death of the deceased by negligence in his treatment of her. There are two statements involved in this. The one is that the treatment of the deceased by the

[3] R *vs* van Schoor, 1948 (4) S.A. 349.

accused with dicumarol, which is the drug he used, caused her death, and the other statement is that he was negligent in so treating her. The evidence seems to show as conclusively as can as a rule be shown in cases of this sort that the deceased died as a result of what is called dicumarol poisoning, which was caused by the quantity of dicumarol she had taken, and that is not contested by the defence. The deceased was a healthy woman; there was no reason why she should have died, but the signs found in her body after death were consistent with death caused by the administration of dicumarol. The only question is whether the accused was negligent in his administration of dicumarol and whether that negligence resulted in her death. So, gentlemen, the main issue before you is, was the accused negligent?

Counsel for the defence yesterday quoted a case,[4] which came before the Privy Council, where it was decided in effect that an accused person was not guilty of criminal negligence unless that negligence was of a gross character. In the course of the judgment in that case the following passages, quoted by counsel for the defence, occur: That a doctor was not criminally responsible for a patient's death unless his negligence or incompetence showed such disregard for life and safety as to amount to a crime against the State; and that gross negligence must be shown before a medical practitioner could be held liable. That case went to the Privy Council from West Africa where the English law applies, and the statement quoted to you by counsel for the defence was a perfectly correct statement of English law. But it is not our law; the basis of our law in this country is the Roman-Dutch Law, and it is not part of our law that a person, whether professional or not, can only be found guilty criminally if he is guilty of gross negligence. In our law the test of negligence is exactly the same in civil as in criminal cases[5]; that is, it makes no difference whether a medical practitioner is sued civilly for damages by a patient who alleges that he has been negligently treated, or is prosecuted criminally by the Crown as in the present case—it makes no difference at all. In our law a man is liable criminally for negligence whether his negligence is gross or slight. So, gentlemen, the quotation put before you by counsel for the defence has absolutely no application to this case and must be disregarded.

Now I must refer to what I think should be considered when you come to discuss the case against the accused. I must first of all deal with the nature of this drug, dicumarol, which was used. It seems to be perfectly clear on the evidence that dicumarol is a dangerous drug. It is a drug which, when administered, lowers the clotting capacity of the blood, to put it in ordinary layman's language, and is indeed used for that purpose; it is used in cases of thrombosis in order to reduce the clotting power of blood; but, if that clotting power is lowered too much, bleeding may result and death may follow

[4] R. *vs* Akerele (1943) 1 All E. R. 367.
[5] R. *vs* Meiring, 1927 A.D., 41.

in consequence of loss of blood. The doctors tell you, however, that it is not a serious danger. The serious danger is that there may be bleeding into the tissues of the brain, and they tell you that if such bleeding is not checked in time it may cause the death of the patient, although this may not happen in a large number of cases. But that, according to the evidence, is what happened in the present case; there was bleeding into the brain tissues which resulted in the death of Mrs. B.

The specialists in their evidence all agree as to the properties of this drug, dicumarol. They go further and say that knowledge of this danger is part of the stock-in-trade of the medical profession, and they tell you that the drug is specially dangerous because its action is variable and its effects upon the individual unpredictable; you can never tell what the effect is going to be on a particular individual. It may act in a certain way upon A and it may act in a totally different way upon B. Not only that, gentlemen, but they tell you that the effects upon a particular individual may vary from time to time. That is the evidence of Dr. Gilroy and Dr. Stephen Lewis, and the evidence of Dr. Jackson and Dr. McLeish is consistent with that. So, because of its possible effects and because of its unpredictable behaviour dicumarol is a dangerous drug. In fact Dr. Stephen Lewis told you that because of its dangerous character dicumarol has now been deleted from the British Pharmacopoeia, and Dr. McLeish said that he now uses the drug much less frequently than before because of its danger. Therefore, gentlemen, the evidence seems clear that dicumarol is a dangerous drug.

As to whether it is common knowledge in the profession, that this is a dangerous drug, Dr. Gilroy and Dr. Stephen Lewis, both lectureres to University students, tell you that medical students at the Universities are taught that dicumarol is a dangerous drug. From those two experts at least one knows that the dangerous properties of this drug are taught to medical students at Universities. They also tell you that general practitioners should know the qualities of the drug, that it is common knowledge, and in fact two general practitioners who were called gave evidence before you that they know of its dangerous qualities. Dr. O'Byrne, who treated Mrs B. on the South Coast of Natal in 1950, told you that he treated her with dicumarol, that he gave her two doses of it, but he says that he watched her and he says he would not have given it to her if there had not been a hospital available where a patient could be kept under observation and could receive the necessary treatment at very short notice. Then, Dr. Beck was asked about his use of the drug and he said he never used it except under the eyes of a specialist and under safeguards, and he told you that he was frightened of the stuff. Even one of the chemists who was called, Mr. Steyn, told you that he would not sell this drug over the counter because he knew from the maker's pamphlet that it was a dangerous drug. So, gentlemen, there is the evidence, that the fact that dicumarol is a dangerous drug is common knowledge in the profession, so that the accused should have had this knowledge.

Then there is evidence as to the precautions that should be taken in administering the drug. A lot of evidence was led on this point, but summarised it amounts to this, that the drug should not be administered unless prothrombin index tests have been taken, not only initially before any dose is administered, but also daily while it is being administered, and, even when it is temporarily left off, in order to give the prothrombin level an opportunity of rising until the effect of the drug has been stabilised; and even after that there should be these prothrombin tests at intervals of two or three days. The doctors, of course, are dealing with ordinary cases and not with cases of emergency in which it may be necessary, as in the case of thrombosis, to give a dose in a hurry without making the prothrombin test. In the ordinary course of treatment the drug is not administered without prothrombin tests being taken in the way I have indicated. Where there are no facilities for these tests to be carried out, then, of course, other precautionary measures must be followed. The next precaution is that while the drug is being administered the patient must be kept under observation and should be seen daily in the initial stages; perhaps not so frequently in the later stages when the effect of the drug has been stabilised. Furthermore, if complications arise, and in particular if bleeding begins to occur, then counter measures must be taken immediately; in the first place the taking of the drug must be stopped and vitamin K must be given and blood transfusions must be done. The doctors tell you that knowledge of these precautionary measures should be possessed by general practitioners, because they are common knowledge in the profession. Dr. Jackson told you that, Dr. McLeish told you that, and the accused did not dispute it. It was put to the accused in cross-examination that you get individual reactions to drugs and that there are contra-indications, that is, indications that the drug should not be used in the case of a particular individual, and the accused agreed with that. He also agreed that complications have occurred in the use of a number of drugs, and he said he had no reason to believe that those factors would not apply in the case of dicumarol; and then he went on to agree with counsel for the Crown that before dicumarol was prescribed the average medical practitioner should know the proper dosage to be administered to patients, that he should look out for contra-indications, that is, signs that the drug should not be used at all in the case of a particular individual, and that he should know what special care and caution should be observed in the event of complications after the drug has been administered. In the case of dicumarol, bleeding is the complication that the medical practitioner should look out for. So the accused does not dispute that knowledge of these matters should be possessed by general practitioners.

That, gentlemen, is the general background of the case. The Crown alleges that accused was negligent in certain respects. If you refer to the sheet containing further particulars you will see that first of all there is a general allegation of negligence. It is said that the accused, a registered medical

practitioner, when prescribing for and treating the patient, failed to exercise that degree of skill, care, diligence and ability which he could reasonably be expected to exercise. That is a general statement of negligence framed in terms of the legal definition I have read to you. Then, in the following paragraphs, (*a*) to (*g*), are given details of the manner in which the Crown says accused generally failed to exercise proper skill, care and diligence in treating the deceased. In dealing with them I will depart slightly from the order in which they are arranged in the document before you. The Crown alleges, first, that accused, before administering and prescribing, failed to acquaint himself with the various strengths or dosages in which the preparation is dispensed, and that he administered the said preparation without any or adequate knowledge of its effects and of its proper administration. Then, in the further particulars it is alleged that he failed to specify in the prescription written out by him the strength or dosage of the preparation to be dispensed to the deceased; that he failed to warn the patient or her husband of the dangerous nature of the drug and of the precautions to be observed; that he failed, in administering the preparation to the deceased, to control the dosage by means of the guidance afforded by a prothrombin index test; that he failed to keep the patient under observation or arrange to have her kept under observation during treatment; and, finally, that he failed to take proper measures when he was informed that the patient was bleeding.

I propose to deal first with the allegation in par. (*b*) of the particulars, namely, that the accused failed to specify in the prescription the strength or dosage of the preparation, because Mr. Pirow's main defence is based on this aspect of the case. In this connection he referred you to the evidence of the accused who said that, when writing out the prescription, he had in mind a dosage of 25 milligrams of the drug to be administered three times a day; and Mr. Pirow referred to the evidence of Dr. Gilroy who said that a dosage of 25 milligrams three times a day would not be expected to harm the average person. Mr. Pirow also referred you to the evidence of Dr. Jackson where he said that a doctor was entitled to rely on the chemist ringing him up if he (the doctor) had omitted to fix the dosage in the prescription. You will remember this evidence. The chemist, seeing that the prescription only referred to tablets without mentioning their strength, without succeeding in making contact with the accused by telephone, looked up the British Pharmacopoeia and on what he found there he issued tablets of 100 milligrams. Counsel for the defence says that the deceased did not die as the result of any dosage prescribed by the accused; she died as the result of the dosage given her by the chemist; the chemist failed to carry out his duty to verify the dosage by asking the accused himself. Hence, he says, the death of the deceased was not due to the accused's negligence but to the action of the chemist in giving tablets of a greater strength than the accused intended to administer. Counsel asked: How can the accused be held responsible for the death of the deceased when it was caused by the administra-

tion of a drug in a strength which he never prescribed. You, gentlemen, must consider that argument and decide whether it is a valid argument or not. There is evidence before you that the prescription was a faulty one; it was negligently made up in two respects, firstly, the dosage to be given to the patient was not stated, and, secondly, the tablets were mentioned without specifying their strength. The evidence of Dr. Jackson and Dr. Stephen Lewis is that the prescription was faulty in those respects. That seems to be a matter of common sense; a doctor should not prescribe for a patient, more especially a dangerous drug, without specifying the dose which the patient is to receive. If the prescription had not been made out in that negligent manner there would have been no opportunity for the chemist to make any mistake; he would have carried out the instructions and there would have been no possibility of mistake. Gentlemen, in cases of negligence —I will try not to make this unnecessarily technical—lawyers draw distinction between negligence which precedes and leads up to an accident and provides the conditions in which the accident occurs as being, what they describe, as a *causa sine qua non*—the cause without which the accident could not have occurred. They draw a distinction between that and negligence which follows such negligence and actually does cause the accident. That is called the *causa causans*, the causative cause. Counsel for the defence does not put his defence precisely on that basis; he does not say that the negligence of his client was the *causa sine qua non* and that the negligence of the chemist was the *causa causans*, but, as I understand it, he is rather inclined to put it on the basis that his client was not negligent at all. You must consider that aspect of the case, whether or not it can be said that *the* cause of the tragedy was the mistake of the chemist and not the negligence of the accused.

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a hypothetical case, assume that a chemist, when he gets a prescription ordering tablets without specifying the strength, does not refer to the British Pharmacopoeia but simply supplies tablets which he has in stock and that they happen to be 100 milligrams; and assume they are administered with fatal results; could the accused in such a case say, 'My negligence did not cause the death; it was the negligence of the chemist in not ringing me up before acting on my prescription?' Well, gentlemen, as men of common sense, you must consider whether an accused person would be able to take that line of defence if the facts were as I have suggested in this hypothetical case. That is what has been suggested to you in the present case. In considering the argument for the defence on this aspect of the case you must bear that in mind.

And, then, gentlemen, there is a further consideration. You have to ask yourselves whether you are prepared to accept the accused's statement that he intended to prescribe 25 milligram tablets. Counsel for the Crown suggested that you should not accept that. On this aspect of the case you must ask yourselves, where could the accused have got this information about the 25 milligram tablets? The expert witnesses were unable to refer you to any publication in which it is suggested that a dosage of 25 milligrams three times a day is efficacious in a particular complaint; it is not mentioned in any textbooks or medical journals so far as they could ascertain. The evidence of the accused, as was pointed out to you, was vague in the extreme on this point. Consider what the accused's knowledge of dicumarol was at the time. He tells you that in some discussion with his colleagues he had heard dicumarol mentioned as an anti-coagulant and that he had some recollection that at that discussion a dosage of 25 milligrams three times a day was mentioned. Then, he says he knew that dicumarol had been beneficial to his mother, but he knew nothing whatever of the dosage that was given to her; all he knew was that she had taken four tablets of the stuff. You must ask yourselves, did he know that there were dicumarol tablets of varying strengths or did he simply know that there were tablets of the preparation. He does not say that he knew they were of varying strengths, and at that stage, as his evidence makes clear, he had no idea that the drug was in any way dangerous. That being the case, was there any reason why he should have formed the intention at that stage of specifying the strength of the tablets? Or did he not intend simply to write tablets because he did not know that the drug was dangerous at all. You must ask yourselves those questions, gentlemen, because if, owing to his lack of knowledge of the properties of this drug, he simply intended to prescribe tablets, then, is not his faulty prescription merely part of his general ignorance as to the proper use of this drug? Those are considerations you should bear in mind. The accused has told you that he definitely had in mind a dosage of 25 milligrams three times a day. Counsel for the Crown has challenged that, and you must decide whether you can accept that statement by the accused.

Assuming that you do accept that the accused intended to prescribe 25 milligrams to be taken three times a day, the question still is whether or not he was negligent in deciding upon that dosage without the precautions which the expert witnesses told you are necessary in the use of this drug. The experts have told you that the drug is dangerous and that precautions are necessary because it is dangerous. They do not say that precautions are necessary only when used in quantities of about 300 to 500 milligrams; not one of them said that; they all said that these precautions, namely, the prothrombin index tests, the regular supervision, are necessary, not in the case of the administration of the drug in that strength; but they are necessary because the drug is a dangerous one, and because of the unpredictable character of the drug. Dr. McLeish in particular told you that precautions were necessary irrespective of the dosage. When Dr. McLeish said that he did not, of course, mean that if you give one milligram you must exercise these precautions; he was speaking of a dosage such as a medical practitioner might prescribe and he would not prescribe one milligram. Now, counsel for the defence, in dealing with that evidence, was inclined to wave it aside, saying in effect that these experts are merely being wise after the event. Well, you have to consider whether that is so or not. Two of them have told you that they teach students at the University. Since students have not begun to practise that is not being wise after the event; it is being wise well before the event. Are you prepared to brush aside that evidence of the experts in that way? And, in any event, whatever decision you come to on this particular aspect of the case, that is, the one connected with the issue of the prescription, that does not conclude the case, because even if you find that the negligence in writing out the prescription did not cause the deceased's death, that that was due to the mistaken action of the chemist rather than the writing out of the prescription, there are still other aspects of the case to be considered, because there are these other grounds of negligence which are alleged against the accused.

The first of these is that the accused prescribed without adequate knowledge. The accused has told you that he had no knowledge of the drug acquired by any instruction he received at University because when he was at the University the drug was not known; it was not at any rate the subject at lectures. But then he tells you further that before he prescribed he read no literature at all on the subject. It is explained that he is a busy practitioner; as he put it to Mr. Justice B.,* his desk was piled high with literature he was never able to read. Gentlemen, that may be so; he may be a busy man; he may have difficulty in finding time to read all the literature that comes to him by way of medical journals, makers' circulars and that sort of thing, but, gentlemen, does that excuse him? I have mentioned to you some of the authorities to the effect that a doctor is expected to have the necessary knowledge, and if, because he is too busy to read the literature

*Mr. Justice B. was the husband of the deceased.

that comes to him, he has not got that knowledge of a particular drug, then the answer is he must not prescribe it. That seems to be the common sense position. The accused tells you that the only information he gained about the properties of the drug dicumarol was from the discussion about which he told you. As I have said, he was vague as to where the consultation took place, or with whom; I think that all he could say was that it took place somewhere on some occasion three or four years ago; he is vague as to the knowledge he gained; he only gained the information that the drug was an anti-coagulant and that the dosage was 25 milligrams at a time. Then, gentlemen, there was the knowledge that it had been used in the case of his mother and had been strikingly beneficial, but he tells you that he gained absolutely no knowledge of the properties of the drug from that administration, except that it had been beneficial in the case of thrombosis, because he was not told and did not know what dosage was used. Well, on that knowledge he prescribed this drug dicumarol, and the question for you is whether that was sufficient. Did he exercise reasonable care in prescribing the drug on that knowledge? Or did he fail to exercise reasonable care?

That is the first ground of negligence alleged in the particulars, and I have dealt with the one relating to the prescription. In regard to the next three, which I summarise, that is, the failure to warn the patient or her husband of the dangerous nature of the drug and precautions, failure to have prothrombin index tests taken and to keep the patient under observation, it seems to me that all those particulars can be taken together because, if you find those were acts of negligence, then they all sprang from the same root cause, namely, the accused's ignorance of the dangerous qualities of the drug and of the means necessary for safeguarding the patient against the dangers. They fall under the general heading of treating the patient without adequate knowledge. I want to mention to you one or two points in regard to these particulars. First of all, in regard to the failure to warn the patient as to the dangers, counsel for the Crown was rather inclined to the view that you should disregard that because the accused had no knowledge of the drug and having no knowledge he could not be expected to warn. That may be so, but still it falls under the general heading of negligence in treating without knowledge. In regard to the necessity of warning the patient as to the dangers, Dr. Jackson told you that if the patient was not in hospital he (Dr. Jackson) would warn the patient to be on the look-out for signs of haemorrhage and if that happened he would advise him to stop taking the drug and report to a doctor. Dr. Jackson's evidence is that unless the patient is in hospital he should be warned. Dr. McLeish told you that, assuming that the deceased had no family doctor in Pretoria but one in Johannesburg, he would have advised her what to do in case bleeding started. He would also have had her transferred to the care of a doctor in Johannesburg and he would not in any case have prescribed the drug without a prothrombin test, that is, he says, irrespective of dosage. So that,

when the accused issued the prescription, you must consider whether it was not his duty to warn the patient of the danger inherent in the use of the drug, that bleeding should be looked out for and what should be done if bleeding started. That is the evidence and you must consider whether he should not have done that. Now, what did he do? As you have heard from the evidence, after seeing Mrs. B. the accused told the Judge that he had given Mrs. B. a prescription, which he handed to the Judge, and told him that it was a drug which thinned out the blood and prevented it from clotting, that Mrs. B. was to take a course of it and come back and see him in three weeks. The witness says that the accused did not say that the drug was dangerous; he did not mention any precautions. That evidence is not challenged in any way by the accused, and it seems to me evidence which you can accept. Then, in regard to the failure to have a prothrombin index test taken and failure to keep the patient under observation, the accused admits he did not do these things because of his ignorance, and he admits that as a general practitioner he should not have been ignorant of it. As I have said, those particulars of negligence seem to fall under the general heading of prescribing and treating without knowledge.

Now, the last one seems to be on a somewhat different footing, namely, the particular where it is said that the accused failed to take proper and adequate measures on being advised that the deceased showed signs of abnormal bleeding. That differs from the others because it does not depend entirely on the accused's initial ignorance of the drug. You will remember the evidence of Mr. Justice B. as to what he did when he learned from Mrs. B. that bleeding was taking place. He said that on the 10th August, that was the Sunday, bleeding, which had begun a day or two before that, became more pronounced and he thereupon telephoned the accused and told him that Mrs. B. was bleeding and 'we were beginning to be concerned', I think, was the expression used. He said that the accused told him that there was no reason to be alarmed, the drug was doing good but she must leave it off, the condition would right itself and bleeding would stop in a day or two. That evidence the accused admitted was substantially correct. In regard to this counsel for the defence has stressed the point that Mr. Justice B. did not tell the accused that bleeding was serious or excessive, and the accused told you that there was no such intimation to him and therefore he did not regard it necessary to take any special precautions or even look up the literature. But, gentlemen, you have got to consider whether this information should not have acted as a warning to him. You will bear in mind that he had not told the Judge that bleeding was a dangerous sign or even that it was to be expected and in the circumstances you have got to consider whether the accused, as a medical practitioner, should not have been put on his guard; whether he should not have realized that this was a warning to which he must pay attention. It must have been quite clear to him from the fact that Judge B. had tele-

phoned him that the Judge attributed some relation of cause and effect, that he connected the bleeding with the administration of the drug. In other words, the accused should have realized that the Judge was of the opinion that this bleeding was due in some way to the drug, and that at least should have been a warning to him. You must consider that. The matter did not rest there. According to Mr. Justice B., in the next two or three days Mrs. B.'s condition got worse and he rang up two or three times with, as he puts it, an increasing note of urgency. Then, when on the 12th, Tuesday, he suggested that Mrs. B. and her daughter, Mrs. M., should go over to Pretoria on the following day, Wednesday, the accused said that was not convenient and an appointment was made for Thursday. Well, on the Wednesday the condition of the deceased was worse; and the accused was notified by Mr. Justice B. that the deceased was too weak to travel to Pretoria, bleeding having increased, and then, according to the Judge, the accused said Mrs. B. must be given some vitamin K tablets. That is admitted by the accused and there is no dispute on that point. Even at that stage the accused did not think it necessary to look up the literature and acquaint himself with the properties of this drug. Then, on the following day, Thursday, vitamin K tablets were ordered and administered. Mrs. M. rang up the accused and asked what the proper dose was and was informed, and, according to Mrs. M., she told the accused that bleeding had increased; there was now blood in the urine and the stools of the deceased, and then the accused said that an intravenous injection would be necessary on some condition as to which there is a dispute. Mrs. M. says that he said if the bleeding became excessive, whereas the accused says he said if the bleeding did not stop. It does not seem to me that the difference is very material. What is material is that on this day, Thursday, the accused, having received the information that bleeding had increased, that there was now bleeding in the urine and the stools, did not even then think is necessary to look up the literature and find out what the properties of this drug were; he says he only did that on the Friday after he had spoken to Dr. Beck, when he looked up some medical book and then found out something as to the properties of this drug.

Gentlemen, it is for you to consider whether, when the accused received information as to the condition of the patient he was negligent in not immediately looking up some literature on the subject, or, if that was not available, whether he should not have got in touch with a specialist or general practitioner with experience and got some information as to the properties of dicumarol. This failure to do anything, the accused tells you, was due to his ignorance of the drug. But as a general practitioner, what should he have done on the Monday, the 11th August, after having received the first telephone call from Mr. Justice B.? Should he not have had a prothrombin index test made and taken steps to keep the patient under regular observation and to administer vitamin K and, if necessary, to cause a

blood transfusion to be done? If he had taken those steps on the Monday, would the patient's life have been saved? You must ask yourselves that question. You have heard the evidence. If you come to the conclusion that there is no reasonable doubt that the deceased's life would have been saved if those steps had been taken and that in fact the accused did not do anything, apart from issuing the prescription, and that his negligence in that regard caused the death of the deceased, then you will find him guilty. If you have a reasonable doubt in this respect, then you cannot find that the accused's negligence resulted in the death of the deceased and he must be acquitted. That is the test on this aspect of the case. Have you or have you not any reasonable doubt as to whether the deceased's life could have been saved if on receiving the information that there was bleeding, the accused had taken steps to acquaint himself with the properties of the drug he had prescribed and with the necessary measures to be taken, and if he had taken steps to put them into effect. On this point you must consider the evidence with regard to the physical condition of the deceased. The evidence is that although she was 70 years old she was in a good physical condition; she had never suffered any serious illness in her life except that on one occasion she had pneumonia or something of that sort. The doctor who held the post-mortem examination told you that there was no organic disease present which was likely to have resulted in her death. The medical experts told you of the measures that should have been taken—a prothrombin index test, constant observation and, if bleeding occurred, the administration of vitamin K and blood transfusion. These are the means by which a patient's life is saved if bleeding occurs, and they are effective. Dr. Gilroy told you that if these precautions are taken, then the cases where death occurs are very rare.

I have tried to put the main features of the case before you, gentlemen. You must direct your mind to the question whether there is any reasonable doubt on the question whether the death of the deceased would have followed if the accused had taken the necessary steps immediately upon being informed that bleeding had started. If you have no reasonable doubt that he was negligent in that regard you will find him guilty; if you have any reasonable doubt on the question you will give the benefit of such doubt to the accused and acquit him.

Will you now please consider your verdict.

The jury, after having retired and considered their verdict for 25 minutes, returned a unanimous verdict of guilty.

After hearing the evidence of Mr. Justice B. in mitigation and counsel for the defence, Mr. Justice Roper passed the following sentence:

Sentence

Roper, J.: T. v d M., the jury unanimously found you guilty of culpable homicide, and I am required to pass sentence upon you. This is a painful

case, because it is dreadful for a medical man to find himself in the position in which you find yourself to-day. I take into account the consequences which this conviction will have upon you; it will probably have unfortunate results upon your practice. And I take into account the fact that you must have passed through a painful and harassing time between the deceased's death and the conclusion of this trial. Mr. Justice B. has gone into the witness box and given evidence in mitigation. He does not desire, nor does any member of his family desire, to take up a vindictive attitude as far as punishment is concerned, nor would it be fitting for the Court to take up such an attitude. But I have to consider the sentence in relation to the gravity of the offence. Your counsel has suggested that this is more in the nature of an unfortunate accident than anything else. I think possibly in a sense it is so, because I think that, not knowing much about this drug, you—if I may use the expression—took a chance in administering it. Still, it was negligence, and the Court cannot pass over lightly negligence in the case of a man in the medical profession, who takes upon himself duties which involve life and death. In all the circumstances of the case I sentence you to a *fine of seventy-five pounds or three months imprisonment with hard labour.*

Dicoumarol: Pharmacological and Toxicological Aspects*

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Dicoumarol is the active substance in spoiled sweet clover, the ingestion of which causes 'haemorrhagic disease' in cattle, a condition characterized by marked increase in blood clotting time. It was introduced in 1941 for prevention and treatment of diseases associated with thrombosis (i.e. clot formation), e.g. coronary thrombosis, thrombophlebitis, etc. The drug produces effects following oral administration and was therefore suggested as a substitute for heparin, which is given parenterally.

Blood clotting is a complex phenomenon not completely elucidated; coagulation depends upon the formation in the liver of prothrombin. This is converted by enzyme action into thrombin which combines with fibrinogen present in the blood to form the fibrin base of the blood clot.

Heparin prevents the conversion of prothrombin into thrombin. The extent of its effect when administered may be gauged by the clotting time *in vitro*. This can be ascertained by simple procedures at the bedside. Dicoumarol delays clotting by preventing the formation of prothrombin in the liver. The extent of effect can be checked by estimation of the 'prothrombin index', a measure of the amount of prothrombin activity in the blood. This requires 'the services of skilled and experienced laboratory personnel[13], i.e. 'a well-trained technician able to reproduce results on successive determinations with the same plasma and thromboplastin, under the direction of an experienced clinical pathologist.'[38, 45]

Dicoumarol is administered orally. Absorption from the gastro-intestinal tract is incomplete and inconstant.[14, 34, 35] The drug combines with plasma proteins and accumulates in the tissues.[34] The rate of transformation varies but is generally slow; it depends to some extent on the dose given. Small doses are absorbed and metabolized rapidly.[34] The products of metabolism have not been identified. Excretion through the renal system is slow and erratic.[34]

The circulating blood contains a certain amount of prothrombin, hence the effect of the drug will be delayed until this amount has been utilized, i.e. 12-48 hours.

The response to dicoumarol therapy therefore varies in individuals and

* The spelling followed here is that adopted in the British Pharmacopoeia.

in the same individual depending upon circumstances.[14, 24, 25, 29, 35, 46-50] The effects of the drug are prolonged for varying periods (2-10 days or longer) after administration has been stopped.

The margin of safety between the therapeutic dose and toxic amounts is narrow.[7] It is not possible to give a dose schedule which will be suitable for every patient.[32, 35, 44, 49] The scheme of dosage generally used gives a high initial dose of 300 mg. followed by 200 mg. the next day and maintenance dosage thereafter to keep the prothrombin index at a safe level. The dose required varies from 25-100 mg. daily and is determined by the prothrombin index for the day. It is therefore essential for the prothrombin content of the blood to be estimated each day. Some clinicians recommend that the drug should not be used unless this can be done.[7, 8, 13, 14, 18, 21, 22, 25-28, 33-35, 43, 47-52, 54, 59] Many physicians consider that the treatment should be carried out in hospital.[8, 10, 26, 34, 48, 49, 52, 53, 55, 59] It has been stated as an 'invariable rule', that no dicoumarol shall be given unless the prothrombin index data for the day are available.[8, 35, 38] No aspirin, salicylates, or sulphonamide may be given.[28, 29]

It has been stated that 'there is no such thing as an average patient where dicoumarol therapy is concerned'.[33]

Clinical opinions vary regarding the optimum safe levels for the prothrombin index; 20-40% appears to be the most acceptable range of values. It should be noted that there is no correlation between 'safe levels' and clinical or toxic effects.[37, 42] Toxic effects may occur in spite of careful observation and dosage to provide 'safe levels'.[14, 15, 21, 25, 28, 29, 32, 34, 37, 39, 41-43, 45, 46, 50, 53, 58]

Toxic effects are due to excessive lowering of the prothrombin levels in the circulation and may be the result of hypersensitivity, accumulation or excessive dosage of the drug.[13, 17, 35] Coagulation of the blood is greatly decreased and haemorrhages occur in all tissues and from all orifices, generally beginning with epistaxis and haematuria and frequently purpuric skin rashes and, in severe and fatal cases, including haematemesis[43] and haemorrhage into the pericardium and in the brain. The patient must be examined daily and the urine tested, since these effects may occur even though the prothrombin index is within the 'safe' range.[41] The toxic effects appear most often from the sixth to the tenth day of therapy[34] but may be delayed until administration has been stopped.[32]

Haemorrhages with dicoumarol therapy are more severe than with heparin.[55] Cerebral haemorrhage is one of the most common terminations in fatal cases.[7, 8, 25, 26, 58]

The toxic dose of dicoumarol cannot be stated definitely. Considerable variation has been noted in the amounts which have been followed by toxic effects; also in the period of administration of the drug:

A single dose of 300 mg. produced major haemorrhage.
A total dose of 2,500 mg. in 7 days produced haematuria.

A total dose of 2,800 mg. in 32 days produced purpura.
A total dose of 2,500 mg. in 10 days produced a fatal result.
A total dose of 2,000 mg. in 11 days produced a fatal result.
A total dose of 2,300 mg. in 23 days produced a fatal result.[10, 22, 28, 43]

The following conditions increase the tendency to toxic effects:

1. Pathological conditions in the liver, kidneys, heart, blood vessels or blood.
2. Concurrent administration of aspirin, salicylates or sulphonamides.
3. Age: elderly persons may be more susceptible.

It is essential that:

1. The patient should be under constant supervision.
2. Prothrombin estimations should be carried out daily.
3. Immediate treatment should be instituted in the event of any haemorrhage, however slight.
4. The patient and/or the family should be warned of the dangers of the drug.[14, 38, 47]
5. The drug should not be administered except with a new prescription each time.[47]

The chief contraindication to the use of the drug is lack of facilities for daily prothrombin estimations or lack of proper clinical use of such estimations.[21, 22, 38] The use without such facilities 'cannot be too strongly condemned'.[38]

Treatment of complications of dicoumarol therapy, i.e. of haemorrhage, necessitates:

1. Immediate stoppage of administration.
2. Blood transfusions using fresh (or stored) whole blood, repeated if necessary.[49, 59]
3. Administration of vitamin K or K_1 oxide intravenously.[12, 13, 22, 24, 25, 27, 32, 36, 42, 52, 54, 57, 58] Aqueous vitamin K preparations are not so efficient.

The danger of haemorrhage persists in spite of treatment[35] on account of delayed excretion of the drug and continuance of effects for several days.[7, 12, 17, 24]

Reports show wide variations in the occurrence of toxicity, e.g. in one series of 1,983 cases the occurrence was 5·2%, i.e. of haemorrhage with 2 fatal cases.[12] Other reports[25] give much higher figures for cases of haemorrhage, e.g. up to 28%; 36 deaths[41] due to dicoumarol had been recorded in the literature up to January 1951. The results of a questionnaire sent out to a large group of physicians in America were reported[51] in *Time*, 1952. About 50% of the group (228 in all) reported the incidence of haemorrhagic complications and 28% gave information of 122 deaths. In all cases, the patients had been carefully supervised during treatment.

In the case under consideration, the deceased was a healthy woman of 70 years, who was given dicoumarol on 2 occasions as part of the treatment for recurrent thrombophlebitis.

On the first occasion 2 doses (200 mg. and 100 mg. respectively) were given by a doctor, who (not having facilities for laboratory control and estimation of prothrombin index) was careful to examine the urine repeatedly for any signs of haematuria, and to keep the patient under careful and constant observation. No untoward or ill effects were noted.

The second instance was about 3 months later when the deceased was again given dicoumarol for the same condition, but by another doctor,

under whose instructions she took 3,800 mg. over a period of 10 days with a fatal result after a further 11 days. Epistaxis, haematuria and a petechial rash with haemorrhage from a 'blood blister' in the skin commenced about the eighth day and progressed. Signs of cerebral involvement occurred about the seventeenth day with death from cerebral haemorrhage 4 days later, i.e. on the twenty-first day after the start of treatment. The drug was stopped on the tenth day. Vitamin K tablets to be taken orally were ordered on the fourteenth day by the doctor, but the patient was already showing severe toxic symptoms, and was given repeated blood transfusions and intravenous vitamin K therapy by a group of physicians under whose care she was placed.

The prothrombin index was not estimated at any time until the fifteenth day, i.e. when the patient was under treatment for toxic effects. The figures recorded were:

15 August 1952	±	30%.
16 August 1952		37%.
17 August 1952		21%.
18 August 1952		No value obtainable.
19 August 1952		No value obtainable.
20 August 1952		34%.
21 August 1952		27%.

There were no apparent contraindications to the use of dicoumarol in this case and no indication of hypersensitivity. The post-mortem examination (i.e. analysis) showed traces of dicoumarol in the body.

Important Points

The important features in this case as shown in the evidence given in Court are as follows:

1. The dose of dicoumarol taken daily by the deceased under the doctor's instructions, i.e. 300 mg.

2. The fact that no prothrombin estimations were done until the deceased was under the care of other doctors for toxic effects of the drug.

3. The complete disregard by the doctor of the reports of epistaxis, haematuria, etc., which were made on at least 3 occasions by reliable members of the family.

4. The admission by the doctor of a complete lack of knowledge of the dangers of the drug, its potentialities and its dosage and of the necessity:
   (*a*) For control of the therapy by prothrombin estimations;
   (*b*) For constant supervision of the patient by himself or by another physician;
   (*c*) For warning to be given to the patient and/or her family regarding early toxic effects and the signs thereof.

5. The carelessness shown in the prescription of the drug. This was for 'Tabs. Dicoumarol, 40', with instructions that one tablet was to be taken 3 times daily. No dose was stated. No 'strength' of tablet was quoted.

There appears to be some confusion in regard to 'strength' and 'dosage' of tablets containing drugs. The terms are not synonymous. Tablets are prepared in certain strengths, i.e. to contain stated amounts of the particular drug, for issue of the drug to the patient. Care must be taken to see that the dose or strength of the preparation given does not exceed the current B.P. range of dosage.

In the case under review the pharmacist was unable to contact the doctor and, acting on his own discretion and as noted above, gave 100 mg. tablets. This is well within the B.P. dose range. The only criticism which can be offered, is that 25 mg. or 50 mg. tablets might have been issued in place of 100 mg.

Deaths associated with dicoumarol therapy are due to hypersensitivity, lack of correlation between prothrombin index or toxic effects due to overdosage.

In this particular case there was no evidence of hypersensitivity, or of the prothrombin content of the blood. The fatal result may therefore be attributed to cerebral haemorrhage from overdosage with dicoumarol, no control having been exercised over the treatment.

It is important to note that dicoumarol has been deleted from the 1953 B.P. It is replaced by ethyl-luscoumacetate, i.e. Tromexan, a derivative of dicoumarol with the advantages of quicker onset of action and less tendency for cumulative effects, but requiring the same close supervision and prothrombin estimations, especially as sudden and unpredictable decrease in prothrombin levels may occur with this drug.

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The Objective Detection of Lying

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It has long been known that when the mind seeks to conceal, the body may betray. While a person may, by word, protest innocence, emotional disturbances—the wide eye and the quick pulse of fear and guilt—may suggest a different story. Use of this knowledge is shown in the ancient custom of making an accused person chew a handful of rice or dry bread. If the rice was spat out dry or the bread not swallowed it was regarded as proof of guilt. Behind the magical explanation which primitive communities give to this trial by ordeal lies the empirical truth that a frightened man has a dry mouth. The scientific explanation is that emotional stress arrests the secretion of saliva.

There is a famous story belonging to antiquity of the discovery of a man's guilty secret by observing his emotional reactions.[1] About 250 B.C. the noted physician, Erasistratus, was living at the court of Seleucus Nicator, king of Syria. Seleucus, then an old man, had married a young woman named Stratonice, about the same age as his children by a previous marriage. His eldest son, Antiochus, fell in love with this lovely young stepmother, but pined away in silence rather than reveal his guilty passion. Seleucus became alarmed at his son's condition and consulted many physicians. None could discover the cause of his mysterious malady. Eventually Erasistratus diagnosed the young man's illness by observing that in the presence of Stratonice his skin became hotter, his colour was heightened and his pulse quickened. He then told Seleucus that his son's condition was incurable because it was caused by a love which could never be requited. The king asked who the lady was and Erasistratus said, 'My wife'. Seleucus implored Erasistratus to give up his wife to his son. Erasistratus asked him how he would behave in similar circumstances. The king replied that he would gladly relinquish his own wife to heal his son. Erasistratus then revealed to the king that it was indeed his own wife with whom his son was in love. Seleucus did not try to deny the logic of the position in which he had been placed by Erasistratus' stratagem. He not only gave up his wife but also part of his empire to his son.

In Mediaeval times, although the concept of the essential unity of body and mind, accepted by all the great physicians of antiquity, had been abandoned, use was still made of bodily changes to seek out guilt. It was known that some people have areas of skin which, when pricked, are insensitive and do not bleed. The discovery of such an area, which is common in hysterical persons, was regarded as infallible proof of witchcraft. The subject, it was stated, bore 'Satan's own sign manual' indelibly branded upon the flesh. A profession grew up of prickers, skilled in finding these Devil's marks. They formed a guild and received a good fee for every witch they discovered in this way. One notorious Edinburgh pricker was personally responsible for the death of 400 women.

Another story of this period[2] is told of a nobleman who suspected his wife of infidelity. He confided his suspicions to a friend who put the lady on trial in the following way. At a dinner he sat next to the nobleman's

wife, casually laid his hand upon her wrist and conversed with her. He mentioned the name of the man suspected by her husband, whereupon the lady's pulse immediately quickened. He later brought up the name of the husband, but perceived no similar response. It is said a confession was later elicited.

In the 19th century attempts to measure bodily reactions to guilt scientifically were made by Cesare Lombroso, an Italian medical practitioner who specialized in the study of criminals. One of his methods involved the use of a crude plethysmograph to measure volume changes in the hand. While the accused person's hand was in the plethysmograph, Lombroso would unexpectedly mention the crime of which he was suspected or place a photograph of the victim before his eyes. He claimed that a diminution in volume occurred if the subject was guilty. At about the same time interest was aroused in an instrument known as the psychogalvanometer, which was found to register a decrease, under emotional disturbances, in the resistance that the body offers to the passage of an electric current. This decrease appeared to be related to the increase in sweating of the palms which occurs during emotion.

In 1914 another Italian, Benussi, became interested in the fact that respiratory irregularities accompany emotional stress. He claimed 100% accuracy in the detection of lying by measuring these irregularities. The following year an American psychologist, Marston[3], claimed that when a person lied the systolic blood pressure rose in a characteristic curve. In 1917 he was requested by the United States Government to report on the value of lie detection methods which might be of military use in counter-espionage work. His committee finally rated the Marston blood pressure test as 97% reliable, the Benussi breathing test as 73% reliable and the psychogalvanometer as unreliable.

In 1921 another American worker, Larson, modified the Marston deception test to provide continuous blood pressure and respiratory recordings. He applied the test extensively to criminal cases and claimed that it could be operated successfully by police officials with 90% accuracy.

In 1923 a Russian psychologist, Luria, demonstrated that the normal smoothness of motor actions was disrupted if the subject was under stress. He devised an instrument for recording this. Some American workers incorporated Luria's 'tremograph' into their lie detectors.

In 1925 the fruit of all this work—the Leonarde Keeler polygraph—made its appearance. Most modern lie-detecting instruments are patterned on it. It gives a continuous record on a moving strip of paper of changes in the blood pressure and in the rate and the depth of respiration.

While all this work on physiological methods of lie detection was accumulating, investigations were also being made along psychological lines. This work produced the word association test, where the suspect was given a list of words to read and told to give his association to each word. The list contained certain key words which could cause distress only to someone

involved in the crime being investigated. If the subject took a long time to produce an association, or an unusually short time, and the association was an unusual one, he was considered to be guilty.

Another method was to make the subject drowsy by the intravenous injection of a hypnotic drug, e.g. scopolamine, or one of the barbiturates. When inhibition was thus removed the subject was questioned about the crime.

There are many difficulties about the drug method.

(i) If the subject has decided to conceal the truth no drug can prevent him from doing so, despite all the publicity given to 'truth drugs'; (ii) The procedure is complicated technically; (iii) The subject does not remember what he said while drugged so is hardly likely to produce a confession afterwards.

These psychological methods are not as reliable as the blood pressure and respiratory tests but are sometimes used as supplementary to them.

The Leonarde Keeler lie detector has been widely adopted by the police in the United States, where it is used as follows:

The consent of the suspect is obtained by telling him that refusal will be construed as guilt. The test is conducted in a quiet room. The subject is seated comfortably with the instrument and the examiner behind him and a blank wall in front of him. The instrument is a portable one and the record is traced with inked pens. The respiratory tracing is recorded by pressure changes from a concertina tube placed round the subject's chest. Systolic blood pressure and individual pulse beats are recorded by inflating a blood pressure cuff round the subject's arm. The cuff is usually inflated to a pressure slightly above that of the diastolic level. Changes in blood pressure are transmitted to a diaphragm which activates the recording pen.

A preliminary record is taken before any questions are asked. This accustoms the subject to the procedure and allows the examiner to exclude any gross circulatory or respiratory abnormality. After this the examiner talks to the suspect using some such formula as:

'This instrument to which you are attached is the well-known lie detector, which has been used successfully for many years for detecting guilt or innocence, and I'm sure it will not fail in your case. Now sit as quietly as possible and just answer my questions "Yes" or "No" '.

The blood pressure cuff is then inflated and a series of 7 to 10 questions asked. The subject must answer the questions because some people attempt to trick the lie detector by concentrating throughout the test on extraneous matters and so failing to react to relevant questions.

Relevant and irrelevant questions are mingled, though the first 2 or 3 questions are always irrelevant. The following is an example of a questionnaire:

Is your name John Smith?
Do you smoke?
Did you have breakfast to-day?
Did you kill Frank White?
Were you born in this town?
Did you hit Frank White with a hammer?

The questions are spaced regularly with a pause of about 30 seconds after each. After a rest, during which the cuff is released, the same questions are put again. This procedure is repeated 2 or 3 times. If the polygraph tracing is equivocal during these tests, another series of questions is asked. These include a larger proportion of questions that could only be of significance to a guilty man, with mention of specific places, objects or times which the police know about the crime.

A suspect is considered to be guilty if, at a relevant question, the blood pressure rises sharply and respiration becomes shallow and is followed by unusually deep breathing. Changes in respiration are not, however, considered essential for concluding that the subject is lying, if the blood pressure change is striking.

If an unusually smooth record is obtained, the subject is not cleared immediately but is given a 'numbers test'. Ten cards with numbers on them are shown to him and he is told to pick one and to lie deliberately when asked which is his chosen number.

If this lie is accompanied by blood pressure change the smooth record of his previous test is considered to be that of innocence. If not, he is considered to be one of those people who have no guilt sense about lying and show no emotional disturbances. In these cases the police tell the suspect that the polygraph shows he is guilty, hoping to win a confession from him.

There are many modifications of the test. One is suddenly to confront the subject while under test with a weapon, forged cheque or other crime exhibit.

The whole aim of the test at present is to break the morale of the suspect in the hope that he will confess. This is necessary because, even in a gadget-minded country like the United States, where lie detection enjoys most credence, very few lower and no higher courts accept its findings as evidence.

The strictness of higher courts in the United States about the admissibility of lie detector results as evidence is exemplified by the following case:[4]

'The defendant was charged with murder and from a conviction in the trial court appealed to the District Court of Appeals, Second District, Division 2, California. During the trial a police officer testified to a conversation between himself and the defendant. The officer testified that he told the defendant he had been placed on a lie detector for a test; that having dealt with machinery the major portion of his life he was sure the defendant would understand that a certain motivating force brings a certain effect, provided the machinery is in operating order. He then explained to the defendant the workings of the polygraph or lie detector, that it is a machine manufactured to register reactions, respiratory rate, pulse rate and certain electrical emanations from the body, that it is based on the premise that it takes more effort to state an untruth than it does a truth and that this effort is reflected on a graph. The officer then stated that he asked the defendant certain questions relating to his age and occupation and that the machine indicated that the answers were truthful. The officer then testified that he showed to the defendant 5 knives, one of which was the knife that was removed from the body of the deceased; the operator showed him one knife at a time and asked him if he had ever seen that knife before, and in each instance the defendant stated he never had. When they showed him the knife

that was removed from the body of the victim, however, there was a violent reaction on the graph of the machine, which indicated that his answer was an untruth. This same test was repeated 2 or 3 times, according to the officer. The officer testified that he then said to the defendant: 'Mr. Wochnick, if you can explain these things to me—maybe you have a logical explanation, maybe you have an explanation you have not told me about yet—if you can explain these things to me, I wish you would. Maybe the whole thing is wrong.' The defendant answered: 'Mr. Zander, I cannot explain that'.

The defendant contended that this conversation should not have been admitted in evidence and that by admitting it under the guise of an accusatory statement the prosecution was able to place in evidence the damaging and prejudicial results of the polygraph or so-called lie detector. The defendant argued that, since the results of such a test are not admissible as evidence, it was therefore prejudicial error to admit them indirectly when they are not admissible directly.

The question of the admissibility in evidence of the results of the lie detector test has not been decided in this State, said the District Court of Appeals. The Court then considered a number of cases in which the use of such tests was questioned and concluded that it was in accord with the views expressed in the majority of these cases that 'the systolic blood pressure deception test for determining the truthfulness of testimony has not yet gained such standing and scientific recognition as to justify the admission of expert testimony deduced from tests made under such theory.'

The State argued that the result of the lie detector test was not placed before the jury, that the mere fact that the occasion for the conversation between the defendant and a police officer was the giving of a lie detector test would not require the exclusion of such conversation if it were otherwise admissible. Furthermore, the State argued, the Trial Court specifically instructed the jury not to consider that portion of the conversation relating to the lie detector test as indicating whether or not there was any reaction to any technical test. Despite the instruction of the Trial Court, the District Court of Appeals said, the evidence of the partial results of the lie detector test with respect to the defendant's reaction on being shown the murder weapon was indelibly implanted in the minds of the jurors and could not but have had a prejudicial effect. The admission in evidence of this testimony places before the jury the result of the lie detector test, which in itself is inadmissible. Accordingly the judgment of conviction was reversed and the case remanded to the Trial Court for a new trial—People *v.* Wochnick, 219, P.(2) 70 (Calif., 1950).

The examiners are usually police officials who have specialized in lie detection techniques. It is emphasized that they must be convinced of the ability of their instrument to detect guilt because this conviction is all-important in successfully putting over the test. It is claimed that 60—80% of those subjects indicated as guilty by the lie detection test eventually confess. In some cases 'the effect of pens swinging to every heartbeat and breath often shatters the morale of the guilty,'[5] but other cases need more than this to induce a confession. One of the ways of doing this is to arrange for the subject to see the examiner and the record during the test. The examiner watches the record and whenever a significant change occurs he smiles knowingly to himself. The subject, seeing the smile and recognizing its implications, will often be robbed of his morale and confess.

Lie detector enthusiasts state that their test is not only of value in criminal cases but useful as a crime deterrent in industry. They quote the case[6] of a large chain store that was losing a large amount of money each year in petty thefts by employees. Leonarde Keeler was asked to test all the em-

ployees with his polygraph. The clerks to be tested were assured that the results of the tests would be kept secret and they would not suffer as a result of being found guilty. They were warned, however, that after 6 months the tests would be repeated and if they were then found guilty they would be dismissed. The first test indicated that 76% of the employees were taking money or merchandise. The second test showed that fewer than 3% were doing so. The reasonable explanation here is that the prestige of the lie detector had frightened most of the culprits into honesty.

The lie detection test is based on the assumption that if a relatively normal person tells an untruth the emotional stress engendered by this violation of his moral code will show itself in circulatory and respiratory disturbances which are recorded and interpreted with scientific detachment.

It will be seen from the description of a lie detection test that it is a stressful experience, whether one is guilty or innocent. The subject is not only submitted to unfamiliar and possibly uncomfortable medical apparatus but everything possible is deliberately done to break his morale. The technique, in fact, becomes an undesirable part of the paraphernalia of detection.

The police justify this use of the instrument by arguing that it helps to gain confessions and so shortens the time and money spent on investigating a crime and at times brings a culprit to book where a crime might have remained unsolved. It may also help to weed out the innocent from among a group of suspects.

Nevertheless this propaganda use of the instrument makes any scientific appraisal of its intrinsic value extremely difficult. There is the fact that a high percentage of successes is claimed. There is also the fact that the people who use the instrument are experienced enough to be aware of its more obvious limitations. They know, for example, that some people will be unsuitable for testing because of gross circulatory disease (such as auricular fibrillation or hypertension) or gross respiratory disease (such as emphysema). They know, too, that results are impossible where a subject is too feeble-minded or mentally deranged to understand what is going on. They know that psychopaths have no moral sense about lying and so will not register any emotional reaction. They are aware of the problem of the phlegmatic type of person who shows very little disturbance and the emotional, anxious type who shows a great deal. With regard to this last problem the police say that a nervous person will react less with each successive test, while a stable but guilty person will react progressively more. They are probably not sufficiently aware of the extraordinary range of variation amongst human beings, whether this results from vasomotor lability or personality differences.

The following assessment of the value of the polygraph, which shows a retreat from the earlier enthusiasm in the United States, is interesting as

coming from a body of great scientific standing—the Atomic Energy Commission.[7]

'The Atomic Energy Commission has established a new and restricted policy on the use of the polygraph, or lie detector, as a tool in its security programme. Under the new policy, the present use of the lie detector at Oak Ridge, where it has been employed in periodic examinations of several thousand employees in security sensitive portions of the manufacturing plants and the commission offices, will be stopped. At the same time, the commission has provided for the use of the lie detector in specific cases of security interest at any AEC installation but on a voluntary basis and on specific authorization, case by case, by the general manager. The lie detector has been used in the atomic energy programme only at Oak Ridge, where it was employed under the Manhattan Engineer District in 1946, and continued under the AEC management since 1947. On the basis of study of the use of the lie detector at Oak Ridge and in other Federal agencies, the AEC has concluded that the machine's techniques offer only indeterminate marginal increase in security beyond that afforded by established and extensive AEC security measures and procedures. It was also concluded by the Commission that the substantial cost of the Oak Ridge lie detector programme in dollars, plus the intangible cost in employee morale, personal recruitment, and labour relations, that might accrue from use of the machine, substantially outweighed the limited advantage of its use. The study showed that there is little data available indicating that the machine has any value in detection of intent to commit sabotage or espionage, or sympathy with subversive movements or ideologies. Its principal value was found to be in detection of pilferage.'

Perhaps the whole subject is best placed in its proper perspective by remembering that all the lie detector does is to measure at 2 points the generalized physiological reaction to stress. It does this by combining 2 simple techniques which have been used by the medical profession for decades. There is nothing magical about the instrument and to trade on the fear and the ignorance of suspects by assuring them that the lie detector never lies because it is some mysterious and infallible invention of science is a reprehensible way of dealing with a person whose guilt is in doubt. All that one can say definitely is that an emotional reaction is a physiological event. It is a non-specific event: there is no special reaction peculiar to guilt. The very term lie detector is a misnomer, perhaps a deliberate misnomer to help the purposes of the police. A better term would be stress detector—and to detect stress is all that the 'lie' detector can honestly claim to do.

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Finger-Prints and Twins

With Special Reference to Identical Twins

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The papillary ridges on the palmar surfaces of the fingers, first described by Purkenje, in 1823, in a University thesis, form patterns which differ from finger to finger, as well as from the fingers of one individual to those of another. The accidental imprint of these patterns on a smooth surface by means of the oily secretion of the skin, or their intentional recording with the aid of an inky substance, is popularly known to-day, especially through its association with the detection of crime, as finger-prints.

So characteristic of an individual are his finger patterns that if a criminal should leave behind him on the scene of his crime his plainly written signature, it would not be a more decisive mark of his identity. Every man possesses, in fact, at his finger ends, a natural equipment of personal seals that he might set upon a document as a sure sign—his sign-manual, ever-ready, inalienable, peculiarly his own; a gauge of identity more difficult to forge than a written signature, simpler, and unvarying over the whole period of his lifetime.

The persistence of the papillary patterns is remarkable. They survive the death of their owner and remain as long as the skin is preserved. The patterns on the fingers of Egyptian mummies, and of stuffed apes, have been found intact. It was this quality of relative permanence that moved Sir Francis Galton, who made the first considerable studies on finger-prints, about 1892, to comment on the Biblical story of the wicked Jezebel who, cast from an upper window, was devoured by dogs so completely that no man might say, 'This is Jezebel'. 'For they found no more of her' the Bible records, 'than the skull and the feet and the palms of her hands'. She might, nevertheless, have been identified, remarks Galton, by her finger-prints, if any record of them had been taken in her lifetime. It may be noted here that palm and sole patterns, as well as those on the pads of the digits, are peculiar to the individual.

Galton's studies led directly to the use of finger-prints in criminal investigation. It is illustrative of the scientific catholicity that was common

before the present century that two such men as Sir William Herschel the astronomer and Sir Francis Galton the anthropologist, eminent in widely different scientific fields, should have been the founders of the practical application of finger-prints to individual identification. Herschel, as chief administrator of the Hooghly district in Bengal, in the middle of the 19th century, first brought the method of finger-prints into regular official employment, in order to obviate the difficulty he found in obtaining credence to the signatures of natives affixed to documents for registration. Galton became interested in Herschel's scheme for classifying finger-prints and, with that passion for precise measurement that characterized his long life of original research, made investigations on his own account, besides getting information direct from Herschel. In 1892 he published his first book on the subject. Three years later, in his *Finger-print Directories*, he dealt fully with the classification of finger-prints and proposed a systematic filing of the finger-prints of prisoners, with a view to their subsequent identification. Shortly afterwards his plan was made the subject of a Government inquiry, with the result that the Scotland Yard scheme used to-day is substantially that proposed by Galton in 1895.

Galton's 3 main types of pattern will be obvious immediately even by a cursory examination of a few hands—the *whorl*, the *loop* and the *arch*

Fig. 1A. Arch (Index Finger).

Fig. 1B. Whorl (Middle Finger).

Fig. 1C. Loop (Thumb).

(Fig. 1). The names indicate the shapes. The whorl consists of concentric circles or spirals; the loop is slanting, and opens to one side or the other of the finger; the arch may be high, 'tented' or flattened. There are, at a closer view, a certain number of transition forms between these; but the 3 main types of pattern are widely distributed and easily discernible.

Any one of these types may be found on any of the 10 fingers; but some fingers favour one pattern more or less than the other patterns. Thus the 'tented' arch is extremely rare on the thumb. Complete symmetry of distribution on the 2 hands of the same person is also rare, and when it does occur is almost invariably associated with all loops, or all whorls, on all the fingers. The forefinger and thumb are least often symmetrical, the little fingers most often.

The commonest of all patterns is the ulnar loop which opens down-

wards towards the thumb side of the hand. The radial loop, opening downwards to the little finger side, is common only on the index finger.

Galton devised the following simple system of symbols:

a = the arch.
w = the whorl.
l = the loop on any finger but the index.
r = the radial loop on the index finger.
u = the ulnar loop on the index finger.

The order in which the fingers are named is:

Index, middle, ring finger of the right hand.
Index, middle, ring finger of the left hand.
Thumb and little finger of the right hand.
Thumb and little finger of the left hand.

So a person whose fingers showed all loops might have the formula:

ull, ull; ll, ll.
or
rll, rll; ll, ll.

Galton's investigations showed 6% of people of the former type and 3.5% of the latter. Persons whose finger patterns were all whorls (represented by the formula www, www; ww, ww) were only 2.5% of his total sample. The remaining formulae were numerous; but none occurred more frequently than twice in 100 cases.

This classification is obviously not sufficient to distinguish one individual from another; but it formed a good basis for division. Actually in some one or other of the 10 fingers of any person the pattern will be found to contain some well-marked peculiarity sufficient to distinguish any 2 persons having the same formula. This in itself would give adequate evidence of identity, because the possible number of peculiarities is very great and any one of them may occur on any of the 10 fingers, giving permutations equal to factorial 10, or 3,628,800. Multiply this number by the number of the possible peculiarities and the product will be a number of types so great that the possibility of mistaken identity, by nature's duplication of a type, is well-nigh infinitesimal.

But the system used by the police authorities is even more proof against the confusion of chance resemblances. The loops, whorls and arches are made up of a number of nearly parallel ridges. At some point near the edge of the pattern these ridges diverge, forming, in the case of loops and whorls, a delta. The number of ridges, counting in a straight line from the delta to the core of the pattern in each finger, is noted; so, in addition to the formal description of the pattern, it is possible to have a series of 10 numbers which may vary from 1 or 2 to 30, each representing a single finger. The actual plan adopted by Scotland Yard to meet the exigencies of indexing and ready accessibility to files is a modification of these 2 principles.

It may be seen how valuable are these minutiae for individual identification; but for comparing groups of people it is practicable to use only the gross patterns. Curious about the possible differences to be found in the

finger-prints of different social classes, Galton examined specimens yielded by statesmen, workmen, idiots and eminent thinkers, and found no difference in the distribution of the patterns. The application of the test to racial groups, however, revealed a suggestive difference. The most uncommon of the 3 patterns is the arch. Galton found it on 13.6% of English right fore-fingers, but on only 7.9% of those of London Jews. More recently Prof. Kristine Bonnevie of Oslo University, in an examination of the finger-prints of 24,518 persons of 4 different racial groups, noted wide differences in the distribution of the patterns as between Norwegians and Japanese.

This strong suggestion of a racial character in finger patterns implies some genetical factor or factors in their formation. On this supposition Galton compared the finger-prints of close relatives and, though the special significance of 'identical' twins was not known in his day, examined those of a few pairs of twins. His investigations supported the supposition, though not in a very striking manner. This is not altogether surprising in the light of the now generally accepted view that the factors determining the patterns are not entirely hereditary ones.

The present writer, 20 years ago, employed the finger-print test (after consultation with, and technical advice kindly given by, Chief Inspector Battley, then head of the Finger-print Department at Scotland Yard) on some 2,000 London school children and used it as a supplementary means of determining monozygosity in twins.

From the total of 424 twin pairs, boy pairs, girl pairs and mixed pairs, it was required to separate the 'identical' pairs. The unlike-sex, or mixed, pairs being removed as unquestionably dizygotic, there remained 280 like-sex pairs, varying in resemblance from 'like as two peas' to complete contrasts. Of these, 165 were set down, after inspection, as obviously non-identical, the physical characteristics of facial resemblance, physique, colour of hair, skin and eyes, configuration of the outer ear and of the teeth, being taken into account. There remained (as judged by closeness of resemblance having regard to these characteristics):

I. The almost certainly identical;
II. The probably identical; and
III. The possibly identical.

These were divided into the 3 groups indicated. The application of the finger-print test to all the like-sex pairs enabled 2 clear-cut classes to be formed: the 'M' Class (presumably monozygotic) and the 'D' Class (presumably dizygotic).

Table 1 shows the entire group of twins of like-sex divided into sub-groups I, II, III and IV, representing diminishing grades of similarity independent of finger-print pattern. The finger-print distribution of the individuals in each of these sub-groups is also given in Table 1 which is divided into 4 sections. The bottom section on the left hand contains

TABLE 1: RELATION OF FINGERPRINT SIMILARITY TO GENERAL RESEMBLANCE IN TWINS

	Number of Fingerprints Similar										
	0	1	2	3	4	5	6	7	8	9	10
I. Identical	0	0	1	0	3	10	17	18	19	13	
II. Probably Identical	0	0	0	0	3	1	4	4	1	3	
III. Possibly Identical	0	1	1	2	3	2	3	2 **M**	2	1	
IV. Non-identical	0	12	11	20 **D**	31	28	29	20	11	3	0

the 'D' Class consisting of those with less than 6 similar finger patterns on the same hand and a complex of striking physical dissimilarities in other particulars. (Six finger pattern similarities were chosen as an arbitrary dividing line). The top section on the right-hand side contains the 'M' Class, all of whom display more than 6 finger-print similarities and, in other physical criteria, a uniformity of twin similarity that is somewhat less. It is justifiable to suppose that the 'D' Class will be composed *exclusively* of dizygotic pairs, and that the 'M' Class will contain a very high proportion of monozygotics.

Table 1 was constructed for a practical purpose; but its theoretical implications are obvious. The high proportion of similarity in the identical groups and of dissimilarity in the non-identical groups gives some indication of a substantial genetic factor in the formation of the patterns of the papillary ridges on the fingers.

The conjecture that the finger-prints of identical twins might be expected to be identical, even in the gross patterns, is completely disposed of. The common inheritance shared by identical twins does not postulate common patterns on the fingers. Dr. Bonnevie, who has published a series of papers on her finger-print researches, finds that the genetic factors affecting the patterns are probably obscure ones. The patterns begin to form very early in pre-natal life and are complete at birth, remaining unchanged thereafter. The characters affecting them are complicated by their connexion with growth factors affecting the rate and manner of the branching, in the embryo, of the nerves and blood vessels of the fingers. The types of design are thus the result of different hereditary factors and of other factors constituting the intra-uterine environment.

Some Neglected Aspects of Barbiturate Therapy*

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London

The more dramatic actions of barbiturates are common knowledge, but there are certain effects these drugs exert which have received only scant attention. The parent drug of all barbiturates is barbitone, the diethyl salt of barbituric acid introduced into clinical practice in 1903 under the name Veronal. Since then barbiturates have slowly replaced the Galenical hypnotics. Whether this was entirely justified is questionable because in many instances barbiturates in the form of tablets have been prescribed simply for convenience.

Between the time of the introduction of Veronal and the present day about a thousand different derivatives of barbituric acid have been synthesized and described in the chemical literature. Many of these compounds have been submitted to pharmacological study, but much of this work is incomplete, largely due to technical difficulties inseparable from the study of the action of drugs on the central nervous system in animals. Comparable studies in man are likely to be easier in the future by advances in electroencephalography, which is opening up interesting possibilities in experimental medicine. It will be necessary however, for the precise significance of the various waves recorded by the EEG to be determined before full use can be made of this new research tool. Isbell and his co-workers[1] have described increase in wave amplitude and beta wave activity in man in chronic barbiturate intoxication. In the interpretation of these findings it must be remembered that American and British workers are not in full agreement about wave nomenclature.

Not until 17 years after barbiturates were introduced into medicine was mention made in the literature of serious toxic effects on the human subject which might follow indiscriminate use of the drugs. These effects were sufficiently uncommon at the time to provide the subject of a Paris M.D. thesis.[2] Even after the publication of this thesis remarkably few cases of chronic barbiturate poisoning were recorded in the literature until quite recent years. Now it is a condition of increasing clinical importance and few

* Based on an address to the British Association of Forensic Pathologists, 22 November, 1952.

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practitioners have not seen a case. The introduction, in 1936, of Governmental restrictions in Great Britain on the sale of barbiturates, probably reduced the incidence of chronic barbiturate poisoning, but, strangely enough, acute poisoning has increased steadily. Now it is something of a social problem and almost a daily event.

The subjects of chronic barbiturate poisoning in England have been in my experience invariably lonely women usually in comfortable circumstances of the type commonly met in places like Bath or Kensington. In the United States to-day, however, barbiturate addiction and chronic poisoning is widespread in both sexes. Unless chronic barbiturate poisoning is suspected, it may be difficult to recognize, because a patient may not readily divulge her habit or even appreciate that progressive chronic ill health is in any way associated with the drug she is taking. After all, her doctor originally prescribed the tablets! The characteristic symptoms of chronic poisoning are chronic lassitude, a peculiar puffy, earthy complexion and hypochromic anaemia. A diagnosis of myxoedema or menopausal changes comes to mind, but treatment for these is of no avail. One of my patients admitted she slept badly and a hypnotic was prescribed; that she took Medinal regularly slipped out when she asked whether she should continue taking tablets ordered some years before.

The more dramatic toxic and side-effects of barbiturates have been fully described in the past 10 or 15 years and a substantial literature is accumulating. Apart from the mental changes, the commonest toxic effect is a morbilliform rash which sometimes spreads into the mouth. The curious thing about the development of this rash is that it does not necessarily occur after prolonged medication; only a few weeks of barbiturate treatment may lead to trouble. Experience shows that the long-acting barbiturates, particularly phenobarbitone, tend to produce skin rashes more readily than the short-acting compounds. A distinction between the rash due to a barbiturate and other causes of peculiar rashes may be made by its morbilliform nature. This is due to the malpighian layer being affected. It is the epidermal layers which are affected by superficial irritants. A few cases of neuritis and blindness have been recorded, but these must be rare.

It is the mental changes accompanying barbiturate poisoning which really cause the greatest concern. Under somewhat artificial conditions these changes have been studied experimentally in man.[3] Volunteers were given large and prolonged medication with Seconal (5-allyl-5-(1-methylbutyl)-barbituric acid), Pentobarbitone and Amytal (5-*iso*-amyl-5-ethylbarbituric acid). The daily dose ranged from 1·5 to 3·8 gm. of the various compounds which were administered for 100 days or more. Of particular interest is the fact that no rashes or blood dyscrasias were produced. The mental changes were studied using psychiatric methods, but the results may be summarized by remarking that they were those which normally might be expected in man in a chronic state of alcoholic intoxication. The paper describing this work

should be read in the original to realize the sort of things that can happen.

Of more immediate importance in every-day practice, also of considerable medical and legal significance, is the 'hangover' which may follow the exhibition of barbiturates. The most striking example of this 'hangover' is after Thiopentone as an anaesthetic for short operations in out-patients. This compound combines with body fat and is subsequently slowly released to produce the untoward effects.[4] Barran,[5] while studying minimal dose effects, showed that mental clarity after consciousness returns is not regained until 45—60 minutes later, irrespective of dosage. Another curious effect of single small or several small doses of barbiturates taken for hypnotic purposes is that such an amount may be sufficient to cloud mental judgment without producing a true hypnotic action. The patient then decides that a further dose is necessary, quite forgetting that some of the drug has already been swallowed. A further large amount is taken, possibly with disastrous effects. Some of these patients may be potential suicides. The late Sir Bernard Spilsbury used to speak of the rehearsals that some individuals might have, and actually kill themselves before they intended to do so.

The foregoing side-effects of barbiturates must be distinguished clearly from the gross toxic actions the drugs exert on the central nervous system.

In a summary it appears safe to classify chronic barbiturate poisoning into 2 distinct groups:

(*a*) Mental obfuscation; and

(*b*) Physical changes due to addiction.

The time of the onset of symptoms relative to the amount of drug taken is of importance in medico-legal practice because prolonged medication with ordinary therapeutic doses does not necessarily cause any of the symptoms described. For instance, a female took 0·12 gm. of Butobarbitone nightly for 15 years without ill effects, and another took 0·2 gm. of phenobarbitone daily for 2 years. Further, many epileptics are given large doses of phenobarbitone and Rutonal (5-phenyl-5 methylbarbituric acid) successfully for long periods. Rutonal, however, in time may cause hypochromic anaemia, which may not be spotted until it is well advanced. The fact that epileptics can usually take these potentially dangerous compounds for long periods without ill effect is a further example of the clinical experience that patients suffering from a disease often tolerate a drug prescribed for it in doses which would give rise to serious side-effects in normal people. The toleration of chemical compounds used in medicine is curiously patchy. For instance, phenothiazine derivatives are in common use, but remarkably few instances of marrow damage have been recorded, yet such might be expected from the known action of phenothiazine itself on haemopoiesis. The precipitating factor in the production of side-effects may be unrecognized. Photosensitivity in sulphonamide therapy was not appreciated for a long time.

There are many instances of pregnant women taking barbiturates to excess. One took 0·2 gm. of Butobarbitone together with 0·01 gm. of pheno-

barbitone daily for 4 weeks; another took 0·2 gm. of Butobarbitone nightly for 2 months; another took 0·015 gm. of phenobarbitone for 6 weeks—at the end of which time she was found to have a total white count of 73,500 per c.mm., of which 32% were eosinophils and 40% lymphocytes. In this last instance it is suggested that the concomitant administration of an antihistaminic may have been a causal factor in the production of marrow changes not commonly observed in ordinary barbiturate intoxication.

The abrupt withdrawal of anything up to 1 gm. of a barbiturate regularly taken daily may precipitate alarming convulsions and delirium. Even the cessation of smaller doses may cause weakness and tremors. In my own experience tapering is always advisable when drug addicts are being treated.

The toxic doses of barbiturates are generally agreed to be 5—10 times the therapeutic dose. Fifteen times a therapeutic dose is usually fatal.

The amount of drug which may be taken at a single dose with recovery appears to vary, however, with the compounds used. The following data collected from various sources illustrates the point. Recovery has followed:

5 gm. Allonal.
3·5 gm. Dial.
10 gm. Phenobarbitone.
10 gm. Evipan.

Butobarbitone, so popular as a general purpose hypnotic, for some reason seems to be better tolerated than other barbiturates and few cases of fatal poisoning following its use are on record. Twenty-four tablets, i.e. 2·4 gm., have produced sleep for 48 hours without ill-effect, and 10·0 gm. (taken by a potential suicide) was not lethal.

In considering the possible toxic actions of the numerous barbiturates available, to say that such and such a number of tablets can produce ill-effects may be misleading. Probably a better approach to the problem in the future will be to consider the significance of the concentration of barbituric acid actually attained in the blood of a patient. All barbiturates used in medicine are broken down in the body to barbituric acid; 15 mg. of barbituric acid per 100 c.c. blood seems to be the maximum amount which can be tolerated without endangering life.

The period of unconsciousness following an overdose of barbiturates obviously must be governed to some extent by the amount of drug actually absorbed; 145 hours is the longest period of unconsciousness so far recorded with recovery. This, curiously enough, followed self-administration of no more than 3 gm. of Allonal.

The treatment of barbiturate poisoning is undergoing changes. Until recently the principle has been to remove as much unabsorbed poison by gastric lavage and then to stimulate the patient by every means available. The drug of choice as a stimulant has been picrotoxin which is 30 times more active than nikethamide and leptazol but slower in action. On the face of it, it must be remembered that the patient is suffering from an overdose of

an anaesthetic and until the anaesthetic can be destroyed in the body or excreted, unconsciousness will persist. Stimulant treatment can only last for short periods before the underlying narcosis reasserts itself. Nillson[6] has, by recent work, demonstrated that much better results may be achieved in the treatment of barbiturate poisoning by appreciating that the patient is in fact over-anaesthetized, and should be treated as such. The basis of Nillson's treatment is the maintenance of a clear air-way, if necessary by bronchial suction, and the judicious administration of fluids intravenously to combat circulatory collapse.

In the Appendix I is a list of some trade names in common use with their chemical composition.

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Appendix I

Trade Name	*Accepted Name*	*Chemical Names*
Alurate	Aprobarbital (N.N.R.)	5-Allyl-5-*iso*propyl-barbituric acid
Amycal, Amytal	Amobarbital (N.N.R.), Amylobarbitone (B.P.C.)	5-*iso*-Amyl-5-ethyl-barbituric acid
Barbenyl, Barbiphenyl	Phenobarbitone (B.P.)	5-Phenyl-5-ethyl-barbituric acid
Bibarbityl	Pentobarbitone sodium (B.P.) and phenobarbitone sodium (B.P.)	Sodium 5-ethyl-5-(1-methyl-butyl)-barbiturate and sodium 5-phenyl-5-ethyl-barbiturate
Butisol sodium	Butabarbital (N.N.R.)	Sodium 5-ethyl-5-*sec.*-butylbarbiturate
Butylone	—	Sodium ethylbutylmethyl-barbiturate
Citopan	Hexobarbitone sodium (B.P.)	Sodium 5-Δ^1-*cyclo*-hexenyl-5-methyl-N-methylbarbiturate
Cyclodorm	Cyclobarbitone (B.P.C.)	5-Δ^1-*cyclo*-Hexenyl-5-ethylbarbituric acid
Cyclonal sodium, Cyclural	Hexobarbitone sodium (B.P.)	Sodium 5-Δ^1-*cyclo*-hexenyl-5-methyl-N-methylbarbiturate
Delvinal sodium	Vinbarbital sodium (N.N.R.)	Sodium-5-ethyl-5-(1-methyl-1-butenyl)-barbiturate
Dial	Allobarbitone (B.P.C.)	5 : 5-Diallylbarbituric acid
Dorico	Hexobarbitone sodium (B.P.)	Sodium 5-Δ^1-*cyclo*-hexenyl-5-methyl-N-methyl-barbiturate
Dormital	Phenobarbitone (B.P.)	5-Phenyl-5-ethylbarbituric acid
Dormonal	Barbitone (B.P.)	5 : 5-Diethyl-barbituric acid
Dormytal	Amobarbital (N.N.R.), Amylobarbitone (B.P.C.)	5-*iso*-Amyl-5-ethylbarbituric acid
Embinal	Barbitone sodium (B.P.)	Sodium 5 : 5-diethylbarbiturate
Embryl, Etoval	Butobarbitone (B.P.C.)	5-*n*-Butyl-5-ethylbarbituric acid

Trade Name	*Accepted Name*	*Chemical Names*
Evipal sodium Evipan sodium	Hexobarbitone sodium (B.P.)	Sodium 5-Δ^1-*cyclo*-hexenyl-5-methyl-N-methylbarbiturate
Euneryl	Phenobarbitone (B.P.)	5-Phenyl-5-ethyl-barbituric acid
Eunoctal	Amobarbital (N.N.R.) Amylobarbitone (B.P.C.)	5-*iso*-Amyl-5-ethylbarbituric acid
Fenatyl	Barbitone (B.P.)	5 : 5-Diethylbarbituric acid
Gardenal	Phenobarbitone (B.P.)	5-Phenyl-5-ethylbarbituric acid
Hebaral sodium	Hexethal sodium (N.N.R.)	Sodium 5-*n*-hexyl-5-ethylbarbiturate
Hexanastab Hexopal	Hexobarbitone sodium (B.P.)	Sodium 5-Δ^1-*cyclo*-hexenyl-5-methyl-N-methylbarbiturate
Hypnogen	Barbitone (B.P.)	5 : 5-Diethylbarbiturate
Intraval sodium	Thiopentone sodium (B.P.)	Sodium 5-ethyl-5-(1-methylbutyl)-thiobarbiturate
Ipral sodium	Probarbital sodium (N.N.R.)	Sodium 5-ethyl-5-*iso*-propyl-barbiturate
Isonal	Mephobarbital (N.N.R.) Methylphenobarbitone (B.P.)	N-Methyl-5-phenyl-5-ethyl-barbituric acid
Kemithal sodium	Thialbarbitone sodium	Sodium 5-Δ^2-*cyclo*-hexenyl-5-5 allyl-2-thiobarbiturate
Luminal Malonal	Phenobarbitone (B.P.)	5-Phenyl-5-ethylbarbituric acid
Mebaral	Mephobarbital (N.N.R.) Methylphenobarbitone (B.P.)	N-Methyl-5-phenyl-5-ethylbarbituric acid
Medidorm	Hexobarbitone sodium (B.P.)	Sodium 5-Δ^1-*cyclo*-hexenyl-5-methyl-N-methylbarbiturate
Medinal	Barbitone sodium (B.P.)	Sodium 5 : 5-diethylbarbiturate
Medipan	Hexobarbitone sodium (B.P.)	Sodium 5-Δ^1-*cyclo*-hexenyl-5-methyl-N-methylbarbiturate
Medomin	—	*cyclo*-Heptenylethylbarbituric acid
Meridol	Mephobarbital (N.N.R.) Methylphenobarbitone (B.P.)	N-Methyl-5-phenyl-5-ethylbarbituric acid
Namuron	Cyclobarbitone (B.P.C.)	5-Δ^1-*cyclo*-Hexenyl-5-ethylbarbituric acid
Napental	Pentobarbitone sodium (B.P.)	Sodium 5-ethyl-5-(1-methylbutyl)-barbiturate
Narcovene Narkotal	—	Sodium 5-*iso*-propyl-5-(b-bromallyl)-N-methylbarbiturate
Nembutal	Pentobarbitone sodium (B.P.)	Sodium 5-ethyl-5-(1-methylbutyl)-barbiturate
Neonal	Butobarbitone (B.P.C.)	5-*n*-Butyl-5-ethylbarbituric acid
Nesdonal	Thiopentone sodium (B.P.)	Sodium 5-ethyl-5-(1-methylbutyl)-thiobarbiturate
Neurobarb	Phenobarbitone (B.P.)	5-Phenyl-5-ethylbarbituric acid
Noctal	—	5-*iso*-Propyl-5-(b-bromallyl)-barbituric acid
Noctinal	Butabarbital sodium (N.N.R.)	Sodium 5-ethyl-5-*sec*.-butylbarbiturate
Normanox	Cyclobarbitone (B.P.C.)	5-Δ^1-*cyclo*-Hexenyl-5-ethylbarbituric acid
Nostal	—	5-*iso*-Propyl-5-(β-bromallyl)-barbituric acid
Nunal	Phenobarbitone (B.P.)	5-Phenyl-5-ethylbarbituric acid
Ortal sodium	Hexethal sodium (N.N.R.)	Sodium 5-*n*-hexyl-5-ethylbarbiturate
Palinum	Cyclobarbitone (B.P.C.)	5-Δ^1-*cyclo*-Hexenyl-5-ethyl-barbituric acid
Palipan	Hexobarbitone sodium (B.P.)	Sodium 5-Δ^1-*cyclo*-hexenyl-5-methyl-N-methylbarbiturate
Pentone	Pentobarbitone sodium (B.P.)	Sodium 5-ethyl-5-(1-methylbutyl)-barbiturate
Pentothal sodium	Thiopentone sodium (B.P.)	Sodium 5-ethyl-5-(1-methylbutyl)-thiobarbiturate
Pentyl	Pentobarbitone sodium (B.P.)	Sodium 5-ethyl-5-(1-methylbutyl)-barbiturate

Trade Name	*Accepted Name*	*Chemical Names*
Pernocton, Pernoston	—	5-(2-Bromallyl)-5-*sec.*-butylbarbituric acid
Phanodorm, Phanodorn	Cyclobarbitone (B.P.C.)	5-$\triangle^1$-*cyclo*-Hexenyl-5-ethylbarbituric acid
Phenobal, Phenornyl	Phenobarbitone (B.P.)	5-Phenyl-5-ethylbarbituric acid
Privenal	Hexobarbitone sodium (B.P.)	Sodium 5-$\triangle^1$-*cyclo*-hexenyl-5-methyl-N-methylbarbiturate
Prodorm	Cyclobarbitone (B.P.C.)	5-$\triangle^{-1}$cyclo-Hexenyl-5-ethylbarbituric acid
Prodormal	Pentobarbitone sodium (B.P.)	Sodium 5-ethyl-5-(1-methylbutyl)-barbiturate
Prominal, Promitone	Mephobarbital (N.N.R.), Methylphenobarbitone (B.P.)	N-Methyl-5-phenyl-5-ethylbarbituric acid
Rectidon	—	5-(β-Bromallyl)-5-(1-methylbutyl)-barbituric acid
Rutonal	—	5-Phenyl-5-methylbarbituric acid
Rymon	Phenobarbitone (B.P.)	5-Phenyl-5-ethylbarbituric acid
Sagatal	Pentobarbitone sodium (B.P.)	Sodium 5-ethyl-5-(1-methylbutyl)-barbiturate
Sandoptal	Allylbarbituric acid (N.N.R.)	5-Allyl-5-*iso*-butylbarbituric acid
Seconal sodium	Quinalbarbitone sodium (B.P.), Secobarbital (N.N.R.)	Sodium 5-allyl-5-(1-methylbutyl)-barbituric acid
Sigmodal	—	5-(β-Bromallyl)-5-(1-methylbutyl)-barbituric acid
Somnifaine	—	Diethylamine salts of 5 : 5-diethylbarbituric acid and 5-allyl-5-*iso*-propylbarbituric acid
Somonal	Phenobarbitone (B.P.)	5-Phenyl-5-ethylbarbituric acid
Soneryl	Butobarbitone (B.P.C.)	5-*n*-Butyl-5-ethylbarbituric acid
Soprinal	Barbitone sodium (B.P.)	Sodium 5 : 5-diethylbarbiturate
Sovinal	Phenobarbitone (B.P.)	5-Phenyl-5-ethylbarbituric acid
Surital sodium	Thiamylal sodium (N.N.R.)	Sodium 5-allyl-5-(1-methylbutyl)-2-thiobarbiturate
Thioethamyl	—	Sodium 5-*iso*-amyl-5-ethyl-2-thiobarbiturate
Thionarcon	—	Sodium 5-ethyl-5-[(butylmercapto)-methyl]-2-thiobarbiturate
Thiothal, Tiopan, Tio-pentamal	Thiopentone sodium (B.P.)	Sodium 5-ethyl-5-(1-methylbutyl)-thiobarbiturate
Tuinal	Amylobarbitone (B.P.C.) and Quinalbarbitone (B.P.)	5-*iso*-Amyl-5-ethylbarbituric acid and 5-allyl-5-(1-methylbutyl)-barbituric acid
Twin-Barb	Butabarbital sodium (N.N.R.) and Pentobarbitone sodium (B.P.)	Sodium 5-ethyl-5-*sec.*-butylbarbiturate and sodium 5-ethyl-5-(1-methylbutyl)-barbiturate
Uronal	Barbitone (B.P.)	5 : 5-Diethylbarbituric acid
Venesetic	—	Sodium 5-*iso*-amyl-5-ethyl-2-thiobarbiturate
Verodorm	Barbitone (B.P.)	5 : 5-Diethylbarbituric acid
Veronal	Barbitone (B.P.)	5 : 5-Diethylbarbituric acid

B.P. = British Pharmacopoeia 1953.
B.P.C. = British Pharmaceutical Codex 1949.
N.N.R. = New & Non-Official Remedies 1952.

The Problem of Race Determination

Limiting Factors in the Identification of the South African Races

The Physical Anthropologist's Approach*

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The purpose of this paper is to stress some scientific aspects of the problem created by the co-existence in one territory of different racial groups. It is only in modern times, as Lord Bryce has emphasized, that race has assumed importance in the minds of people. Wars have been fought for plunder, for land, for conquest and subjugation, for religion, for commercial supremacy, but practically never because of racial unity, racial purity or racial antipathy. The fact that the idea of race has gained a grip on the minds of people and political leaders establishes a duty for the physical anthropologist to elucidate the bases on which the concept of race is built. His task is made more urgent when strong emotional reactions are aroused and differential legislation is engendered by the race problem.

As the question of race has come to loom so large over South Africa, it is essential that we have a clear conception of the meaning of race before we can validly address ourselves to the legal and moral aspects.

What is understood by 'race'? The term is borrowed from biology, not from sociology. In biology, we speak of races when a species of animals or plants is divided into groups or populations which are united by heredity. The members of such a group habitually interbreed; they possess an historical continuity in reproducing individuals of a special appearance. Representatives of race A could hybridize and thus exchange hereditary equipment (genes) with race B but, as the races are disposed in their natural environments at any period, gene exchange *between* the races is minimal and gene exchange *within* each race is maximal.

With no less assurance may be speak of races of Man. The human species is divided into a large number of populations, each of which is united by heredity, the members of which habitually interbreed with one another and

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are historically continuous with earlier populations of specific appearance. There is a tendency for gene exchange to occur chiefly within the race, but interbreeding between races is possible, just as it is among other animals and plants.

Race is therefore a biological concept; it brings in its wake no necessary socio-cultural consequences. But socio-cultural factors—such as warfare and conquest, slavery and concubinage, taboos against extra-tribal marriage, improved means of transport and migration—may influence the relative proportions of intra- and inter-racial exchange of genes. We shall see how the biological outlines of race have in fact become hazy with the progress of civilization and the enhancement of socio-cultural influences in the human polity.

The Races of Man

With this definition before us, let us see how physical anthropologists have classified Man. Strictly speaking, every rural community, every city populace, might be classed as a different race, since gene exchange within such units is maximal. It would, however, serve no useful purpose to classify races in this way. On the contrary, the function of a classification is to introduce simplicity into supposed complexity, order into apparent disorder; the criterion of a good classification is its usefulness to those engaged in the field. Classification is of its essence arbitrary, although all classifications strive to reflect the natural relationships of the subjects classified.

Anthropologists have therefore grouped all the hundreds of human populations into a limited number of major stocks or races. They recognize firstly a white or Caucasoid race, possessing *inter alia* wavy hair, narrow, high-bridged noses, vertical profiles and a skin colour ranging from white in the North of Europe to olive in Southern Europe and brown in India. The Caucasoids are sub-divided into several groups—the Nordic, Alpine, Mediterranean and Armenoid of Northern, Central, Southern and South-eastern Europe respectively. Sometimes the term 'race' is applied to each of these sub-divisions of the Caucasoid, e.g. the Nordic race or the Mediterranean race. This nomenclature may still be useful in parts of Europe, but, in other parts of the world, with great settled populations of white people, it is becoming increasingly difficult to sort out the exact racial sub-groupings which have contributed to the white populations. Thus, in Southern Africa, it would be more useful to speak of the Caucasoid race as the source of our white inhabitants. The Alpine and other sub-divisions would then become 'racial variants', 'sub-races' or, simply, 'physical types'.

A second major human stock comprises the Mongoloid or yellow peoples ranging through vast areas of Asia as (*a*) the Mongolian Mongoloids of Tibet, China, Japan and the Eastern Republics of the Soviet Union and as (*b*) the Malaysian Mongoloids of the Malay Peninsula and South-eastern Asia generally. In addition, (*c*) the Mongoloid race forms the basis of the

Eskimos and of the American Indians, although in the latter, the yellow skin coloration has been replaced by a coppery or red hue.

Thirdly, the Negro or black peoples rank as a major sub-division of mankind, inhabiting Africa and, through ancient migrations, Melanesia. As slave immigrants, this race has given rise to the Negro population of North America.

A less clearly defined group are the yellow-skinned Bush peoples of Southern Africa, whose dwindling descendants, the Bushmen and Hottentots, have some features in common with the Negro race but differ from the Negro in a great number of other features. The Bush physical type is so distinct and goes so much further back in our fossil record than the Negro that it seems we should regard this once widespread group as a distinct Bush race.

Finally, the Aborigines of Australia may form a separate race, though their credentials for race distinctness are more equivocal than those of the other races mentioned.

The Bases of Classification

When we think of a Mongoloid individual, we conjure up an impression of a yellow complexion, slanting eyes with overhanging eye-fold, a broad face, high cheek bones, scanty facial hair distributed in a circular fashion around the mouth and straight, coarse, black hair. In other words, the variations upon which this classification are based have mainly to do with appearance and, especially, the appearance of the face. We recognize our acquaintances and relatives by facial features. It is common knowledge that the face betrays a wealth of information about every individual: from it we glean clues about the race, age, sex, family relationships, state of health and even disposition and habits. Raymond Mortimer, in his *Essay on Clothes*, makes the point neatly: 'If we really had modesty, it is our faces we should conceal. By comparison, our legs are anonymous, our bellies uneventful.'

Apart from the face, stature is useful in classifying the various forms of Man. The range is from the Congo Pygmies (average 143 cm. or 56.3 inches) to the tall Dinkas of the Nilotic Sudan (average 180 cm. or 70.9 inches).[1]

More recently, a series of genetic factors, such as the genes for various blood groups and for taste ability, have been used as aids in classifying Man.[2] This genetic method of classification, it has been claimed, will supersede the traditional methods since, by specifying the gene frequencies in a racial group, more objective assessments of racial affinity become possible. It is interesting to note, however, that the new method has produced a classification virtually identical with that arrived at by the older method. This suggests that the distribution of purely morphological features on which the older classifications are based approximates to the relative fre-

quencies of the more precisely identified genes, which provide the new classificatory basis. A synthesis between the two techniques is clearly needed and this is to be sought in the genetic analysis of the traditional morphological characters. We urgently require more controlled studies on the mode of inheritance of such anthropological features as skin, eye and hair colour, hair form and distribution, eye-folds, prognathism (or jaw protrusion). When sufficient data have been accumulated, it will be possible to define races in terms not only of the genes for blood groups and taste ability, but also of the genes for those visible morphological features which have long been the main objects of study of physical anthropologists.

It is clear that our classification makes use of predominantly *physical* (including physiological) features; *mental* characteristics play no rôle in helping the anthropologist to classify the types of Man. That mental differences may parallel the physical differences is a definite possibility, but we do not yet possess adequate techniques for separating off those mental differences which are due to the impact of the environment from those which are inborn or hereditary or racial. Researches on identical twins are helping to elucidate which mental traits are hereditary, but generalization at this stage would be premature. Biesheuvel's studies on the aptitudes of Africans have led him to this interim conclusion:[3]

'It cannot be stated with scientific certainty that the difference (in mean intelligence test performance) would disappear if environmental conditions were made entirely equivalent for the two races in Africa. Races have developed by a process of genetic differentiation, and in so far as intelligence itself is dependent on genetic constitution, it cannot be excluded from the operation of evolutionary laws. It may turn out that the intellectual differences between races are a matter of "bent" towards particular abilities, rather than of general intellectual capacity; there may be a real difference in the mean level that can be achieved; or there may be no difference at, neither qualitative nor quantitative, given adequate opportunities for development. Only further research can decide between these possibilities.'

It is customary to ascribe personality traits to peoples, especially to *national* groups (which are by no means co-extensive with racial groups). It is common practice to speak of the 'phlegmatic Englishman', the 'sensuous Frenchman', the 'loquacious Welshman', the 'stolid German'. Such personality traits may indeed characterize many members of the several nations but we have no evidence that these qualities are inherent in the genetic constitution and do not result from social behaviour patterns. Still less is it possible to demonstrate personality traits extending over whole races (e.g. Caucasoids, Mongoloids), as distinct from national characteristics.

In short, mental characteristics cannot in any way assist the classifying of the races at this stage of our knowledge.

The Search for Pure Races

Hitherto, we have spoken only of races. In this section, a new idea is raised—namely the purity of races. The question to be asked is: Are the

races, as defined above, pure, or, if not, can they be traced back to pure races?

The traditional approach of physical anthropologists has been to relate the living forms of Man to idealized racial types. If a race of ideal types could be found, it would be a pure race. Thus, consciously or unconsciously, anthropologists have envisaged pure races, existing either to-day or in the past. The quest for pure races has tended to elevate race purity to a sought-after quality. Some anthropologists have, in a measure, been responsible for the over-emphasis on race purity. The extreme example of this tendency is that of the German racist-anthropologists. A few quotations from Günther,[4] who typifies this class of anthropologist, illustrate the lengths to which the ideas of race purity and impurity were carried and their alleged psycho-social consequences:

'Athens sank in the same measure that the blood of her Nordic upper class ran dry.' (P. 168.)

'The Sammite love of fighting, the truthfulness and reserve of the Sabine tribe, and the chastity of its women . . . may well point to these tribes having a strong element of Nordic blood.' (P. 177.)

'Where the Nordics, keeping their racial purity, settle over an unbroken area, some kind of popular government must come into being.' (P. 192.)

'Wealth was a noble thing so long as it was essentially landed property, and belonged to a class that through its race was fitted for ruling, and brought up to own property, and loved wealth not for its own sake, but for the sake of power, and treasure, and honour. Wealth becomes something mean so soon as a class collects it which does not bring a high mind to the task; it becomes something mean in the history of a people under Nordic influence the moment the non-Nordic man comes to riches. He has not inherited the way of life that befits wealth if it is not to be something base. Rule and possession are not in his blood.' (P. 195.)

According to Günther,[5] Alpines are 'petty criminals, small-time swindlers, sneak-thieves and sexual perverts'; Nordics, on the other hand, are 'capable of the nobler crimes'!

The reaction against this one-sided and invalid interpretation of race purity and history has been marked. The extravagant claims of the Nazi racist-politicians evoked replies from Dahlberg, Ashley-Montagu and others and even led the pendulum to swing almost too far in the opposite direction. We find to-day a tendency to deny the validity of the race concept in anthropology. The first draft of the UNESCO statement on race was felt by many to have gone too far and a re-draft has been formulated.

A steadying of the pendulum is required; this will centre on the idea that race in human biology is as useful a concept as it is in other biological fields; but that it brings no known psycho-social and cultural consequences in its wake—and that 'race' does not mean 'pure race'.

We have no evidence that 'pure' races of Man exist anywhere on earth to-day, nor that a 'pure' race has ever existed. Weiner,[6] surveying the developments in physical anthropology since 1935, concludes:

'Prehistoric men (judged from bony remains were quite as variable as modern popu-

lations, so that no evidence emerges from craniometry or osteometry of the former existence of "pure" or homogeneous races.'

Even the rugged-browed Neanderthal Man, who lived in Europe before the end of the last (Würm) glaciation of the Great Ice Age, shows marked variation within his kind.[7] In Southern Africa, we know at least a dozen skulls representing individuals who lived in the Middle Stone Age; yet, even at that remote period, tens if not scores of thousands of years ago, the earliest humans of whom we have record in South Africa are not homogeneous, but differ fundamentally among themselves.[8]

To what depths of time must we plumb to find pure races? The ancient pithecanthropine hominids of Java and China varied among themselves hundreds of thousands of years ago. Of the still earlier South African fossil ape-men, the Australopithecines or Dartians (as Sir Arthur Keith has suggested they be called after their first discoverer, Prof. R. A. Dart), at least 5 or 6 varieties are known from the caves of the Central and Southern Transvaal and the North-eastern Cape Province. Earlier than that, the fossil record does not permit us to probe.

From the dawn of Man in the Pliocene-Pleistocene geological epoch to the present day, we find no trace of a pure race. In the ensuing paragraphs, we shall see how the modern notion of race requires no preconceptions about the present or former existence of 'pure' races.

The Races of Southern Africa

The foregoing concepts may now be applied to Southern Africa. Each of the major groups into which most scientists agree to divide the human species is represented in the sub-continent. Briefly, the segments of the population are:

Negro: About 9,000,000 Bantu-speaking Negroes; the Berg Damaras of South West Africa, about 34,000 Negroes who are not Bantu-speaking, but have adopted the language of the neighbouring Hottentot (Nama) tribes.

Bush: Perhaps 90,000 Hottentots distributed in the Union, South West Africa and the Kalahari; the Bushmen, an unknown number, probably not exceeding 10,000, surviving chiefly in South West Africa and the Kalahari (and Angola).

Caucasoid: About 2,500,000 whites of mixed Caucasoid origin.

About 300,000 Indians, belonging mainly to the Mediterranean branch of the Caucasoid race.

Mongoloid: The Cape Malays, some 35,000 in number, to whose make-up several racial elements have, contributed, although, basically, they belong to the Malaysian branch of the Mongoloid race.

Mixed: About 1,000,000 Coloured folk of varied racial origin (including the Griquas and the Rehoboth Bastards).

The main inhabitants of Southern Africa for the last 300 years have been 3 groups of people, the Europeans, the Bantu-speaking Negroes and the Bush-Hottentot group. The pattern of racial types has, however, been greatly complicated by miscegenation between new settlers and indigenes and between the various hybrid strains which have emerged and which

we have lumped above as the Coloureds. It is well to remember, as Dart[9] has pointed out, that:

'Miscegenation is no new feature in the racial history of Africa. The fossil finds of human bones tell a story of ancient peoples in Africa fusing with one another and with aliens.'

The earliest human inhabitants of Southern Africa are known only by their handiwork, the primitive (Kafuan) pebble tools and later (Chellean-Acheulian) hand axes. Possible clues to the appearance of the makers of these implements are provided by a fragment of jaw bone from the Cave of Hearths, Makapansgat Valley,[10] and the fossil human skull remains from Hopefield near Saldanha Bay. The discovery of these remains by Mr. Keith Jolly and Dr. Ronald Singer was recently announced by Prof. M. R. Drennan.

The first group well represented in the fossil record is the large-headed Boskop race, called after the first fossil human skull discovered in South Africa, found at Boskop near Potchefstroom in 1913.

The next group to appear on the scene were the Bushmen, a race retaining primitive and foetal features in its make-up. It is customary to regard the Bushmen as invaders into the Southern domains of the Boskop folk,[11, 12] but there is no good reason to debar the Bushmen from having taken origin in Southern Africa, as a mutant offshoot of Boskop Man. Hybridization between the Bushman upstarts and the Boskop indigenes—perhaps complicated by later invasions from the Mediterranean zone—resulted in various blends, the descendants of which we see in the Bushmen and Hottentots of to-day.

The invasion of Southern Africa by Bantu-speaking Negroes took place within the last millennium. We have good evidence[9] that these people originated in the North and pressed down the African migration routes to the South. Long before they reached the Southern *cul-de-sac* of Africa, the Negroes had felt the impact of Mediterranean Caucasoids and their genetic endowment bore the stamp of Caucasoid admixture. In the South, miscegenation again took place with the already mixed indigenes, so that Bush and Boskop elements entered into the racial composition of the Negroes. The clicks in many of the Bantu languages spoken to-day in South Africa (except Mocambique) are one cultural betrayal of the Bush influence on the Negro.

While the Bantu Negroes were slowly spreading down the south-east coast of Africa, the newly-arrived European (Caucasoid) settlers were radiating from the south-western corner. Fresh miscegenations took place between European and Bushman, European and Hottentot and, later, European and Bantu. Arab and Oriental traders, Malayan and West African Negro slaves, too, cast their genes into the pool over the centuries. The meshwork of the South African races became, in consequence, ever-increasingly entangled, as indeed occurred in most parts of the world.

This comparatively recent period, when the exchange of genes between

races has become a dominant theme in human evolution, succeeds a period when an opposite tendency was manifest. In an earlier era, probably running to hundreds of thousands of years, the human population of the world was broken up into major segments, geographically isolated from one another by deserts—the ice sheets of the Pleistocene glaciations in Europe, or the sandy wastes of the great trans-African Kalahari and Sahara barriers, or the watery deserts of the oceans. Isolated by the natural environment, the different types of Man evolved their distinctive features. It was a phase when human evolution was divergent; groups of men were growing apart in appearance. The process of divergence seems never to have gone so far as to produce 'pure' races; isolation was never so rigorous or so uninterrupted as to wipe out all morphological overlapping in the marginal population groups; but the *motif* of change was diversification.

With the rise of Man's culture, with his newly-developing ability to resist and even master his environment, a reversal of the divergent trend came into being. The beginnings of agricultural economy, the domestication of animals, the discovery of the wheel and of the boat, revolutionized Man's relations with his environment. He became a migrant, a sailor, an invader. Migration, whether over land or over sea, closed the gaps between populations and racial groups; and, as geographical isolation broke down, racial isolation disappeared. Different races came to occupy the same territory and, since the various races are inter-fertile, hybridization was the inevitable result. For some thousands of years, inter-racial hybridization is evident in the fossil or written records of prehistoric and historic Man. With the consequent pooling of genes, the different forms of Man have ceased to diversify and even existing racial outlines are obscured by the rising hybrid populations. Human evolution has become convergent.

Racial Hybridization

Nowhere more strikingly demonstrated than in Southern Africa, the principles of racial hybridization and convergence have here produced a mélange of such complexity that conflicting viewpoints are held on the racial status of many of the folk inhabitating this country. An example is the Korana, variously claimed to represent a pure line traceable to the Middle Stone Age, a Negro-Hottentot hybrid and a Bushman with considerable Boskop and slight Negro admixture.[13-15]

Recent studies made from the Department of Anatomy of the University of the Witwatersrand have revealed the complexity of the racial elements woven into the faces of various South African groups. In 1937 Dart[16] first demonstrated that living Bushmen of the Southern Kalahari were distinguished by two main types of face, one characteristically Bush in type and the other resembling the face shown by Galloway[21] to be typical of the Stone Age Boskop folk; but strange foreign features cropped out as well,

Mediterranean and Armenoid and Mongoloid. Similar variability has been found more recently by Williams[17] and Eriksen[18] in Kung and Heikum Bushmen and by myself in Auen and Naron Bushmen, while Eriksen, following earlier work of Galloway, has recognized Mediterranean, Nordic, Armenoid and Bush facial types liberally distributed among the Negro faces of the Ovambo. Of course, the possibility cannot be ruled out that, e.g. the Mongolian (epicanthic) eye fold may be a parallel pedomorphic specialization in the Bushman. Nevertheless, in the light of our knowledge of racial movements and contacts in prehistoric Africa over some thousands of years, there is scarcely room for doubt that much of the high incidence of these foreign features reflects ancient hybridization, rather than the recent parallel evolution of features already present in existing racial groups.

All this evidence confirms that a search for a 'pure' race in South Africa would be in vain. Physical anthropologists may still speak of the 'typical Negro' or 'typical Bushman' face; but the studies cited reveal that the individual with the 'typical' face is exceptional rather than usual. It is most difficult to find in one individual a combination of all the features, each of which, *on its own*, may be typical of a particular race—features such as the shape of the nose and protrusion of jaws, the skin and hair colour, hair form on head and body, the architecture of the cheek bones and forehead, the body build and stature, and the blood group picture.

As Kroeber has put it, a race is only an average of a large number of individuals. Inter-marriage and chance mutational factors blur the boundary lines and create marginal intergrades between races, so that it becomes impossible to distinguish race with certainty except at the extremes of the range.

Where miscegenation has created a large hybrid population, as in the Coloured folk of South Africa, a relative stability does tend to emerge; for a new race is being born. Keen[19] has claimed that a relatively stabilized Coloured type may be recognized on cranial features, but that a spectrum of variation exists, even within that type. Such a population no longer requires new mixed marriages to maintain itself; marriages between Coloured and Coloured are sufficiently numerous to ensure not only the maintenance of numbers but the natural increase of the Coloured population. It is logical to expect that, as the Coloured population has grown and the size of the marriage isolate or unit has increased, so the proportion of marriages between Coloureds and non-Coloured racial out-groups would have dropped. In fact, in 1946, as Sonnabend and Sofer[20] have shown, fewer than 10% of the marriages potentially productive of Coloured offspring were between Coloureds and others; over 90% were between Coloured and Coloured. Thus the new mixed race is contributing most to its own perpetuation.

Although the Coloured population is emerging as a new race, it intergrades marginally with the various races which have contributed to its making. Marginal intergrading is due to the wide range of variation within

the Coloured people itself and to the small but definite fringe of continued miscegenation between people 'on either side of the line' or who have 'crossed the line'. There are individual Coloured people whom one could not distinguish on their physical appearance from Caucasoid Whites on the one hand and from South African Negroes on the other; while there are individual Whites and Negroes whom one could not distinguish from Coloureds.

The main difference between the Coloureds and other living peoples, as Sonnabend and Sofer[20] have stressed, is that the particular mixture which produced the Coloureds occurred within historical times and is still fresh in human memory. The earlier miscegenations which have given rise to the other groups in our population are shrouded in the distant past and, indeed, many of them took place before the beginnings of recorded history. Further, the Coloureds are still living cheek by jowl with the main racial groups which produced them and, in fact, are still being numerically reinforced from their parent stocks; whereas in the past, hybrid populations have often completely absorbed or supplanted their progenitors.

Race and the Individual

We may conclude from the foregoing that the concept of race is valid as long as we are dealing with groups of people; racial features are the average of a large number of individuals' features. The old search of the anthropologist for 'pure' races has given way to a conception of races as differing among one another in the frequency with which they manifest specific hereditary features, whether physical (like head dimensions) or physiological (like blood groups).

The modern conception of race submerges the individual and obscures his distinguishing marks. This fact is of paramount importance in considering race in relation to the law; for, although differential legislation is for *groups*, it is the *individual* who is involved in legal proceedings arising from such legislation. It is precisely when we consider the individual that the concept of race is liable to break down, perhaps in more instances than those for which it remains valid. A White with a dark complexion may resemble a Coloured or even a light-skinned Bantu Negro more closely than two Caucasoid Whites of Northern and Southern Europe resemble each other. A Coloured man with a light skin and a high-bridged nose may pass as a European more easily than a White who has high cheek bones and dark colouring.

It becomes clear that any attempt to place all individuals in natural racial categories is beset with insuperable difficulties; more, it does violence to the facts of science. While, in times past, racial categories may have been well-nigh (but even then not quite!) water-tight, the march of evolution has ruthlessly erased the frontiers of race. No physical anthropologist

could set himself up as a scientific arbitrator over these boundaries in a multi-racial community like that of South Africa.

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REVIEWS OF BOOKS

MEDICAL JURISPRUDENCE

Medical Jurisprudence. By I. Gordon, M.B., Ch.B., R. Turner, M.B., Ch.B., D.P.H. and T. W. Price, Ph. D., M.A., LL.B. (Cantab.), B.A., LL.B. (South Africa). *With Additional Contributions by* Prof. M. R. Drennan, Dr. G. W. Gale, Prof. J. A. Keen, Dr. G. J. Key and Dr. H. A. Shapiro. (3rd ed., 1953, Royal 8vo., pp. 992, 143 illustrations and 4 colour plates. 75s.). Edinburgh: E. and S. Livingstone Ltd.

Contents: I. The Law of Southern Africa and its Administration. II. Registration and Professional Discipline in the Union of South Africa and South-West Africa. III. Registration and Professional Discipline in Southern Rhodesia. IV. Judicial Powers of Supervision over the Medical Councils. V. Unprofessional Conduct. Offences prescribed by the Medical Councils and the Medical Acts. The ethical and legal aspects of artificial insemination and sterilisation. VI. Contracts and the Practitioner. The contract to treat a patient. Contracts with other practitioners and assistants. The medical practitioner's insurance policies. VII. Delicts and the Practitioner. VIII. Crimes and the Practitioner. IX. The Public Duties of Medical Practitioners. X. Evidence and the Expert Witness. XI. Identity. XII. The Examination of Blood Stains. Blood groups. Disputed parentage. Chances of proving non-paternity. Blood transfusion accidents. XIII. The diagnosis of death. Cooling of the body. Rigor mortis. Cadaveric spasm. Heat rigor. Post-mortem hypostasis. Putrefaction. Estimation of the post-mortem interval. XIV. Necropsy Technique. XV. Deaths from Rapid Anoxia. XVI. Deaths usually initiated by Anoxic Anoxia. Breathing in vitiated atmospheres. Suffocation. Throttling. Strangulation. Hanging. Choking by foreign body impaction. Drowning. XVII. Deaths initiated by Anaemic and Histotoxic Anoxia. Acute carbon monoxide and acute cyanide poisoning. XVIII. Anaesthetic deaths. XIX. Deaths from burns. Deaths from exposures to low and high environmental temperatures. Deaths from electrocution. XX. The Medical Investigation of the Cause of Death. Obscure deaths from natural and non-natural causes. XXI. Deaths from Acute Neurogenic Cardiovascular failure. XXII. Suspected Poisoning. The post-mortem detection of poisons. XXIII. Mechanism of wound production. Abrasions. Bruises. Incised, lacerated, and punctured wounds. Complications of wounds. Primary and secondary shock. Haemorrhage. Infection. Age of wounds. Distinction between ante-mortem and post-mortem wounds. XXIV. Regional Injuries of Medico-Legal Importance. Head injuries. Spinal cord injuries. Injuries to the neck, chest, abdomen, and urogenital tract. Injuries to the limbs. Patterns of injury in traffic accidents. XXV. Firearm Wounds. XXVI. Sexual Offences. XXVII. Abortion. XXVIII. Infanticide and Concealment of Birth. XXIX. Medico-Legal Aspects of Acute Alcoholic Intoxication. XXX. Medico-Legal Aspects of Mental Defect or Disorder. The legal responsibility of the insane. Insanity and divorce. The care, supervision, and treatment of mentally disordered persons. XXXI. Medico-Legal Aspects of Workmen's Compensation in the Union of South Africa. XXXII. The Law relating to Poisons and Habit-forming Drugs in the Union and South-West Africa. XXXIII. The Law relating to Poisons and Dangerous Drugs in Southern Rhodesia.

Anyone acquainted with the second edition of *Forensic Medicine* by Rhodes, Gordon and Turner will agree with the statement in the authors' preface that this is not merely a third edition of, but rather a successor to, that book. The metamorphosis which the work has undergone is not due, in any marked degree, to the increase in medico-legal contributions, but to the fact that a completely new legal section, representing more than one third of the volume, has been added.

Legal Section. This new material satisfies the need which has long existed for a comprehensive and clear statement of the law as it affects the medical practitioner in South Africa. The value of this contribution to our legal literature cannot be doubted and it is, therefore, in no carping spirit that the suggestion is put forward that when the next edition is printed the legal section should appear as a separate book. This will avoid the criticism that the present volume contains much which cannot reasonably be said to fall within the scope of the generally accepted definition of forensic medicine, viz. 'the science concerned with the application of medical knowledge to certain branches of law, both civil and criminal' (Glaister, *Medical Jurisprudence and Toxicology*, 9th ed., p. 8).

The legal section is intended primarily for the general practitioner, and the authors have very wisely, as far as this is possible, avoided juristic quibbles and niceties and presented a straight-forward statement of the law. The wide range of subjects covered will be appreciated by a glance at the Table of Contents, and the medical practitioner can be assured of finding in this book an answer to very nearly every legal problem that he is likely to encounter in his professional career.

Without wishing to detract from the general high standard of the book the following statements appear to be open to comment and perhaps criticism:

P. 10. The general proposition that, if an accused pleads guilty, 'the Court may, if it is satisfied that he fully appreciates what he is saying, proceed to sentence him forthwith' is, of course, only correct in respect of the powers of a Supreme Court. An Inferior Court is bound by the provisions of section 286 of Act 31 of 1917.

P. 51. The authors state: 'In the case of serious crime, such as murder, it is incumbent on the practitioner to give information to the police which he considers necessary. Thus if a practitioner is called upon to attend a person suffering from shot or stab wounds in circumstances which make it clear that the patient has been criminally assaulted or is a fugitive from justice, he must clearly inform the police'. The proposition is so loosely worded that it is difficult to know what principle it is intended to lay down. How, for instance, is the practitioner to decide what is a 'serious crime', and in what way is it 'incumbent' on him to give information. Do the authors suggest this as an ethical or as a legal duty? If a legal duty, then one would welcome a reference to the law which creates such a duty. It is submitted

that in the case of murder there is a clear legal duty on the practitioner to report the death to the magistrate in terms of the Inquest Act or the Births, Marriages and Deaths Registration Act. He need then do nothing further until he is approached by the police and should then give whatever information is required—not merely what 'he considers necessary'.

It is further submitted that the practitioner may be acting improperly if, without consent, he were to inform the police that he had attended a person suffering from shot or stab wounds in circumstances which made it clear that the patient had been criminally assaulted. Surely it is the privilege of the assaulted person to decide whether he desires to lay a criminal charge or not? A husband who has, for instance, been stabbed by his wife might not desire a prosecution and in such circumstances the practitioner has neither an ethical nor a legal duty to inform the police. Moreover, if criminal abortion is a serious crime, and indeed it has always been so considered, then the authors seem guilty of a palpable contradiction by stating at page 739 that the practitioner should never inform the police that a patient has been criminally aborted, 'if she refuses to co-operate'.

P. 51. There would appear to be no authority for the unqualified statement that 'attempted suicide is not a crime in Roman-Dutch law'. In R. *v* Peverett (1940 A.D. 213) the Court intimated that this is a question that has yet to be decided.

P. 68. There is a complete absence of any authority in our law for the authors' assertion that a woman commits adultery if she is artificially inseminated with the semen obtained from a third party donor. It is only an expression of the authors' opinion (which may or may not be correct) and it should have been presented as such.

On the same page appears the categorical statement that it is not illegal to sterilize a patient for the purpose only of preventing him or her from having children. Once again no authority is offered, in support of this proposition. A more acceptable statement of the law on the subject appears at p. 252. Here we are told that to remove tissue from a living person where it is possible that the operation may 'seriously harm or prejudice the person concerned', appears to be contrary to public policy and therefore unlawful. This surely is the correct test to apply in deciding whether sterilization, in the circumstances mentioned, is lawful or not, and if so, is it not at least arguable that a patient who has been sterilized suffers serious 'harm or prejudice'?

P. 159. Dealing with the question of what consent is necessary when an operation on a minor is undertaken, the authors offer the following example. '(7) A baby is brought to hospital. Examination leads to a diagnosis of acute intestinal obstruction. The father of the child refuses to consent to the operation. It is suggested that the duty of the surgeon is to operate.' If by this illustration it is intended to suggest that the practitioner has a legal duty to operate in such circumstances, it is undoubtedly an incorrect statement of the law. Only where the minor 'has no guardian or other person in lawful charge of him from whom it is possible to obtain consent in time for the thing to be done with benefit' would it be lawful for the practitioner to operate without such consent. (Gardiner and Lansdown, *S.A. Criminal Law & Procedure*, 5th ed., p. 94). It would be unlawful to operate when consent has been refused, though it may well be that by refusing to permit the operation the father makes himself liable to a criminal prosecution.

The book is marred in several places by what appears to be hasty writing. At p. 239, for instance, we find this rather extraordinary passage: 'Such an absurd result follows from placing undue weight on statutory law and failing to realize that custom is still the basic source of law'. A statute is a law made by the legislative authority which the Courts must enforce. It is meaningless, therefore, to talk about placing 'undue weight on statutory law'. Further 'the sources of the criminal law of South Africa are the Roman-Dutch law, the law of England, and the enactments of legislative bodies'. (Gardiner and Lansdown, *supra*, p. 4) To refer to custom as 'still the basic source of law', is, to say the least, misleading. This same clouded thinking is noted at p. 252 when it is stated: 'It would appear therefore, to be contrary to public policy, and therefore unlawful'. An act is only unlawful if the law says it is so. In the absence of such a provision in the law an act, however contrary to public policy, is not unlawful.

Medical Section. This is a refreshing contribution to our medico-legal literature, and reflects the salutary influence of the application of sound principles and concepts of modern pathology to forensic problems. This section of the work will be found to have importance and utility far beyond the geographical boundaries of South Africa. A good example of the value of a modern medico-legal contribution is referred to in the editorial pages in this issue.

This third edition will be of great practical value to the medical practitioner and undergraduate preparing himself for a field in which formal instruction is often wholly inadequate. The legal practitioner will find much material with which to equip

himself for cross-examination—often one of the best techniques yet devised for bringing to light the facts of a situation.

DISPOSAL OF THE DEAD

The Disposal of the Dead. By C. J. Polson, M.D., F.R.C.P., Barrister-at-Law, Professor of Forensic Medicine, University of Leeds, R. P. Brittain, M.A., B.Sc., M.B., Ch.B., B.L., LL.B., Senior Lecturer in Forensic Medicine, University of Leeds and J. K. Marshall, M.B., Ch.B., Lecturer in Forensic Medicine, University of Leeds. Edited by C. J. Polson. (Pp. 300. 21s.) London: English Universities Press Limited, 1953.

Contents: Part I. 1. Historical Introduction.
Part II. Mediate Disposal. 2. The Duty to Dispose of the Dead. 3. Anatomical Dissections and Pathological Examinations. 4. Certification of the Cause of Death. 5. The Medical Certificate of Cause of Death. 6. The Certification and Registration of Still Births. 7. Registration of Deaths. 8. The Formalities of Disposal of the Dead. 9. The Duty to Inform the Coroner of a Death.
Part III. Cremation. 10. A Short History of Cremation. 11. Cremation Legislation. 12. Recommendations for Revision of the Cremation Regulations. 13. The Principles and Methods of Cremation. 14. Crematoria.
Part IV. Burial. 15. The Law of Burial. 16. The Place of Burial; Memorials: Modern Cemeteries: Burial Fees. 17. Funeral Rites.
Part V. Exhumation. 18. The Law and Practice of Exhumation.
Part VI. Embalming. 19. A Short History of Ancient Embalming. 20. The object of Modern Embalming. 21. The Changes after Death and their Relation to Embalming. 22. The Circulatory System in Relation to Embalming. 23. Embalming Instruments and Apparatus: Embalming Fluids: Cosmetics. 24. The Modern Practice of Embalming.
Part VII. 25. Funeral Direction. 26. On Coffins. 28. Transport of the Dead. Index.

This new and interesting publication makes it clear that the issues which confront those responsible for disposing of dead bodies are more considerable and multifarious than would appear at first sight. The survey is comparative in its scope; and it is interesting to find that the location of crematoria in various countries is listed, including those under construction. This is an index of the care and thoroughness which has gone into the compilation of this volume.

It is true that the law relating to interference with dead bodies differs materially in different parts of the world, but the excellence of the information which the authors have surveyed undoubtedly makes the volume of considerable value outside the United Kingdom, although the primary emphasis is on English legal practice.

The authors draw attention to the anomalous position in the United Kingdom which makes unlawful all unofficial post-mortem examinations, i.e. those not the concern of the coroner, and made solely for the advancement of knowledge. This unsatisfactory state of affairs until recently existed also in South Africa as Masters and Shapiro pointed out in 1951 (S. Afr. Med. J., **25**, 376). The position, however, was remedied by a recent Act of the Union Parliament. This legislation deserves careful study by those concerned with similar problems in other parts of the world, particularly as the South African legislation aimed to legalize other forms of interference with dead bodies necessary for establishing Eye Banks, Bone Banks, etc.

This book is much more than a survey of formalities and procedures. The historical approach is adopted throughout and this lends interest as well as profit to the account. Much useful information about the pathological changes after death is also included and it would be wise for those concerned with the performance of autopsies to become acquainted with cognate issues, e.g. the problems which face the embalmer. These may be of considerable importance when the medico-legal pathologist is required to handle cases which become the subject of judicial inquiry, only after they have already been embalmed.

Although this volume will be of considerable importance to all those concerned with the disposal of the dead, it should prove of particular interest and value to medical and legal practitioners.

DOCTOR, PATIENT AND THE LAW

Doctor and Patient and the Law. By Louis J. Regan, M.D., LL.B. (Pp. 545. Second edition. £4 9s. 3d.) St. Louis: C. V. Mosby, 1949.

Contents: I. Malpractice or Negligence. 1. Definition of Malpractice. 2. Malpractice Illustrated. 3. Malpractice or Negligence. 4. Chapter Discussion.
II. Physician and Patient. 5. The Legal Duty of a Physician. 6. Prenatal Injuries. 7. Physician-Patient Relation. 8. The Physician is Not an Insurer. 9. Care and Skill Required of a Specialist. 10. Chapter Discussion.
III. Special Rights to be Respected. 11. There Must be Consent for Operation. 12. There Must be Consent to Autopsy. 13. Invasion of the Right of Privacy. 14. Privileged Communications. 15. Chapter Discussion.
IV. Liability for Act of Another. Section 16. Negligence of Partner, Employee, or Associate. 17. Chapter Discussion.
V. Hospitals. 18. Charitable Hospitals. 19. Private Hospitals. 20. Public Hospitals. 21. Hospitals Not Liable for Acts of Attending Physician. 22. Right to Practice in a Hospital. 23. Chapter Discussion.
VI. Expert Witnesses. 24. The Expert Witness. 25. Experts of Different Schools. 26. Hypothetical Questions. 27. Chapter Discussion.
VII. Expert Testimony. 28. Expert Testimony is Usually Necessary. 29. When Expert Testimony is not Necessary. 30. *Res Ipsa Loquitur.* 31. Chapter Discussion.
VIII. When Physician is Liable. 32. Proximate Cause. 33. Contributory Negligence. 34. Statute of Limitations. 35. Chapter Discussion.
IX. Evidence. 36. Evidence—Admissibility. 37. Chapter Discussion.
X. Proof. 38. Proof Required of Negligence. 39. No Liability for Honest Error of Judgment. 40. Admissions. 41. Nonsuit and Directed Verdict. 42. Chapter Discussion.
XI. Miscellaneous. 43. Abortion. 44. Adoption. 45. Adverse Party Called as Witness. 46. Artificial Insemination. 47. Blood Grouping Tests in Affiliation Pro-

ceedings. 48. Contraceptive Statutes. 49. Contracts in Restraint of Trade. 50. Coroners and Medical Examiners. 51. Criminal Malpractice. 52. Damages. 53. Disclosure Under Statute. 54. Dying Declaration. 55. Federal Food, Drug, and Cosmetic Act. 56. Implied Promise to Pay for Professional Services. 57. Insane Persons. 58. Insurance. 59. Licence to Practice. 60. Medical Societies. 61. Narcotics. 62. Pharmacists. 63. Special Defenses. 64. When a Child is Born Alive. 65. Wrongful Death Actions. 66. X-ray Films, Ownership of.

XII. The Dentist. 67. Duties and Liabilities of Dentist Analogous to Those of Physician. 68. Chapter Discussion.

XIII. The Nurse. 69. General Considerations. 70. The Nurse in Industry. 71. The Nurse and Malpractice. 72. Chapter Discussion.

XIV. Forms. 73. Consent to Operation. 74. Consent to Operation upon Minor. 75. Consent for Autopsy. 76. Consent to Disclosure of Privileged Information. 77. Patient Leaving Hospital Against Advice. 78. Acknowledgment of Hazards of Particular Therapy. 79. Obstetrician Unable to be Present at Delivery. 80. Authorization to Treat Patient who has Recently or Partially Aborted. 81. Patient Fails to Carry out Advice. 82. Patient Fails to Keep Appointment. 83. Patient Discharges Physician From Case. 84. Physician Withdraws From Case. 85. Patient Injured, or Alleging Injury, in Hospital.

XV. Malpractice Vulnerability, Self-Test. 86. General Questions. 87. The Physician—Personal Factors. 88. Equipment, Records, and Employees. 89. Self-Determination of Vulnerability.

XVI. Malpractice Prophylaxis. 90. Prevention of Malpractice Claims—Education of the Physician. 91. Prevention of Malpractice Claims—Education of the Public. 92. Malpractice Prophylaxis—Admissions. 93. Malpractice Prophylaxis—Records. 94. Malpractice Prophylaxis—Unethical Criticism. 95. Malpractice Prophylaxis—Tact. 96. Malpractice Prophylaxis—Liability for Act of Another. 97. Malpractice Prophylaxis—Operation, Autopsy, Hazardous Therapy. 98. Malpractice Prophylaxis—X-ray, Diathermy, Etc. 99. Malpractice Prophylaxis—Insufficient Treatment. 100. Malpractice Prophylaxis—Miscellaneous.

XVII. Allegations in Malpractice Suits. 101. Frequent Allegations in Malpractice Suits.

XVIII. Case and Comment. 102. Illustrative Cases with Comments.

XIX. Medical Defense. 103. Strong Defense Necessary.

XX. A Malpractice Program. 104. Why a Malpractice Program. 105. Fundamentals of a Malpractice Program.

Conclusion. Table of Cases Cited. Other Citations and References.

This is undoubtedly one of the most comprehensive surveys of the medico-legal problems which can arise from medical practice.

Not only is almost every conceivable contingency covered, but the wealth of documentation and the illustrative case reports make this one of the most reliable textbooks for consultation and reference.

The chapters dealing with the *Expert Witness* and *Expert Testimony* deserve careful study by all members of the medical profession in particular. In this portion of the book Professor Regan has laid down admirable principles which should guide the medical practitioner when he finds himself in the witness box. No medical graduate should embark upon his career without a careful study and appreciation of the wise counsel set down so systematically and concisely by this experienced author.

Such problems as the moral and legal implications of artificial insemination find very thorough treatment; indeed, Professor Regan's account of the issues at stake in artificial insemination is one of the most complete available and his authoritative and dispassionate survey is typical of the way in which all matters, whether controversial or not, have been handled throughout the volume.

The scope of the book is so considerable that it undoubtedly has a close bearing on the professional problems of practice in all parts of the world.

CRIMINAL AUTOBIOGRAPHY: AN ANTHOLOGY

Men of the Underworld: The Professional Criminals' Own Story. Edited by Charles Hamilton. (Pp. 336 + xvi. 13s. 6d.). London: Victor Gollancz Limited. 1953.

Contents: I. The Reign of Lawlessness. *Adventure of the Drugged Grooms* (Henry Tufts); *Boodles of Queer* (W. Stuart). *Your Money or Your Life* (Captain Lightfoot). *A Foray in Detroit* (Sile Doty). *Gambling on the Mississippi* (George H. Devol).

II. Crime's Golden Age. *Cracking a Burglar-Proof Safe* (Langdon W. Moore). *In Cahoots with the Cops* (George M. Bliss). *How the Bank Sneak Works* (Harry Schindler). *The Terror of Wall Street* (George Bidwell). *Fleecing the Lambs* (John Phillip Quinn). *I was a Pickpocket* (Anonymous). *The Art of Burglary* (Jack Black). *We Rob a Train* (Al Jennings). *The Truth about the Western Outlaw* (Emmett Dalton).

III. Guide to the Underworld. *The Mob and Its Code* (Chic Conwell). *Burglar Cops* (Anonymous). *Cheating the Law* (Chic Conwell). *Underworld Lingo* (Chicago May).

IV. The Modern Freelance. *A Safe-Cracking Spree* (Herbert Emerson Wilson). *Sticking up a Bank* (Ernest Booth). *Forging Ahead* (Roger Benton). *Strange Case of Henrietta* (Joseph 'Yellow Kid' Weil). *I'll Gyp You Every Time* (Anonymous).

V. Racketeering in the Raw. *Bootleg Days* (James Barbican). *Hijacking Booze* (James 'Limey' Spenser). *The Technique of Murder* (Danny Ahearn). *Dope and the Criminal* (Victor F. Nelson). *The Pari-Mutuel System* (Johnny the Guy). *Syndicated Sex* (Anonymous).

VI. Behind Grey Walls. *Cast in a Dungeon* (Stephen Burroughs). *Living with Lice* (William Stuart). *My First Day in Stir* (Donald Lowrie). *I Survived the Torture* (Ed. Morrell). *A Texas Chain-Gang* (Ernest Booth). *Escape from Hell* (Jack Callahan). *The New Prison* (Roger Benton).

VII. The Road Back. *Fighting to Go Straight* (Jack Callahan).

VIII. Postscript for To-morrow. *How to Prevent Crime* (Louie Whitsitt). *Curbing the Confidence Man* (Joseph 'Yellow Kid' Weil). *Let's Legalize Gambling* (Joseph 'Yellow Kid' Weil. *What's Wrong with the Law* (Victor F. Nelson). *The Prison of the Future* (Ed. Morrell).

Glossary. Bibliography. Index.

This is a remarkable volume in many ways. It is an anthology about crime and criminals written by the expert professional thieves and wrongdoers themselves. Clearly the professional specialist (as opposed to the amateur) in this field is of the opinion that crime *does* pay; yet this is a view difficult to reconcile with the incontrovertible fact that much of this anthology reports a change of heart and the seeking for a new and honest way of life.

The book is an important document for the close attention of all students of the social causes of crime, because it covers the whole anti-social gamut comprehensively and historically from the days of the hold-up man and the train robber to the age of syndicated sex and the disturbing

current problem of teenagers and drug addiction.

This history, as it unfolds, contains much that is a cause for consternation; yet it is not without its lighter side. Indeed, the language of the crook of the 19th century revealed very literary and quite polished prose accomplishments which his contemporary counterpart certainly lacks.

The modern criminal has developed techniques for hiding his fingerprints by the use of gloves and has even resorted to plastic surgery in order to make the camera lie. He is in close touch with recent technical developments but the Editor of this volume reveals an uncritical gullibility in only one place (p. 265). He claims that yellow soap concealed in the armpits will induce an artificial fever.

Mr. Hamilton's book is, nevertheless, an important one for those who are concerned to create conditions which will eradicate much of the crime of our times.

A JUDGE'S NOTABLE TRIALS

A Book of Trials. By Sir Travers Humphreys. (Pp. 237 + xxii. 15s.). Melbourne: London: Toronto: William Heinemann Limited, 1953.

Contents: 1. Belt *v.* Lawes. 2. The Colin Campbell Case. 3. On Trial by Jury. 4. Oscar Wilde. 5. Kitty Byron. 6. The Reubens Brothers. 7. The Crippen Case. 8. The Case of the Seddons. 9. Hooley and Bottomley. 10. Rex *v.* George Joseph Smith. 11. D. S. Windell. 12. The Button and Badge Murder. 13. Some Spies—I. 14. Some Spies—II. 15. High Treason—I. 16. High Treason—II. 17. The Great Pearl Case. 18. William Cooper Hobbs. 19. Bywaters and Thompson. 20. Some Solicitors. 21. Some Forgers. 22. Blackmail. 23. The Gutteridge Case. 24. The Chinese Murder. 25. R. V. Benton. 26. The Case of Ernest Brown. 27. R. *v.* James Camb. 28. John George Haigh. 29. Some Observations on the Case of Mrs. Barney.

Sir Travers Humphreys has long adorned the British Bench and his almost unrivalled experience includes some of the biggest trials of his day. In many of them he participated as Counsel or as Judge.

He inclines to the view that the avid contemporary public interest in crime is a post-Victorian phenomenon and he has interesting views on how particular social conditions and industrial developments determine the pattern of crime.

The volume is written engagingly and the learned Judge is often in a position to provide new information about old and celebrated criminal cases. There has, e.g. long been a common belief that in the notorious *Brides in the Bath* case, a plausible theory of how the murders were committed was staged by a dramatic experiment in Court. 'To demonstrate the point a young woman in a bathing-suit entered the bath and although aware of what was going to happen, went under so quickly when a detective gripped her below the knees that she almost drowned in open Court! It was terrifyingly conclusive and the jury needed little head-scratching to bring in their verdict'. Sir Travers dubs this as a farrago of nonsense and indicates what the real basis for this astounding distortion really is.

It was, of course, in the Seddon case that Sir Bernard Spilsbury expressed the uncontradicted view (on pathological evidence), that the death of the victim (Miss Barrow) was due to acute arsenical poisoning. There can be little doubt that Miss Barrow did in fact die from acute arsenical poisoning, but it is extremely doubtful whether Sir Bernard was entitled to express this opinion on the basis of the anatomical and non-toxicological data available to him.

Spilsbury was prepared to admit that his findings were consistent with death due to 'epidemic diarrhoea', but he excluded this because of the state of preservation of the body. That the body can be preserved by the ingestion of a fatal dose of arsenic is probably completely mythical. The observations of Copeman and Kamerman, published in their paper on *Poisoning by Arsenic in South Africa* in the *South African Medical Journal* of 12 October 1940, make it clear that putrefaction proceeds despite fatal arsenical poisoning and that in the few cases where moderate or good preservation of the cadaver was found, the physical conditions of the grave site, the soil drainage, etc. provided an adequate explanation of what was found at exhumation.

The author's recollections of these famous trials will prove of great interest to medico-legal practitioners, especially to those with medical qualifications who are required to give expert testimony. It is interesting that, when he addressed the Medico-Legal Society as President, he chose the cross-examination of Seddon, together with the evidence of Dr. Willcox as 'perfect specimens of the art of advocacy and the proper attitude of an expert scientific witness respectively'. These are available in the *Notable British Trials Series* and will repay careful study by the potential expert witness.

Sir Travers Humphreys' volume undoubtedly provides the reader with a few interesting and instructive hours.